Moos (S.)

INVESTIGATIONS UPON BACTERIAL INVASION OF THE LABYRINTH IN THE COURSE OF MEASLES

BY

S. MOOS, OF HEIDELBERG

Translated by Dr. MAX TOEPLITZ, New York

[Reprinted from the ARCHIVES OF OTOLOGY, Vol. xviii, No. 1, 1889]

INVESTIGATIONS UPON BACTERIAL INVASION OF THE LABYRINTH IN THE COURSE OF MEASLES.

By S. MOOS, of Heidelberg.

(With eleven lithographic illustrations upon plates i.–vi., and a colored plate, table vii., of volume xviii., German edition.)

Translated by Dr. Max Toeplitz, New York.

I.—Pathologico-Anatomical Part.

I RECEIVED two petrous, with notes referring to them, from a friend not residing in Heidelberg. They were taken from a boy, three years old, who was admitted to the hospital on December 12th with morbilli. and died on December 19th.

Post mortem: Catarrhal Pneumonia.

I did not receive any notes with reference to the condition of the auditory organ during life.

Results of Examination of both Petrous Bones.

Both drum-membranes are united to the promontory, lustreless, yellow-red, handles of malleus not visible. Both tubes permeable for a probe of 1 *mm* in diameter, in appearance unchanged. The tympanic cavity is filled by a gelatinous exudation, the mucous membrane being extremely thickened to the extent of 3 *mm* in diameter, enclosing the ossicles; on the left side the changes are more marked than on the right. The gelatinous exudation consists of granu-

Reprinted from the Archives of Otology, Vol. xviii., No. 1, 1889.

lar corpuscles and epithelial detritus. The microscopical examination of the mucous membrane of the labyrinthian wall gives the same result on both sides.

The Mucous Membrane of the Labyrinthian Wall. Microscopical Examination.

The deeper stratum, the periosteum proper, was hyperplastic, and penetrating largely into the neighboring bone, which thus presented a sinuous appearance. The proliferated periosteum had in places cut off the bone, so that in the centre of the hyperplastic tissue osseous discs partly roundish, partly oval, were visible.

The proliferated periosteum, devoid of cells, was in many portions covered by isolated or grouped colloid spherical bodies. The stratum covering the proliferated periosteum was distinguished by its bright appearance. A delicate reticulum of spindle-shaped cells and thin fibres of connective tissue form their basis; in the lacunæ of the reticulum numerous large yellow, homogeneous colloid bodies could be seen, isolated or in groups; also large round, oval, and pear-shaped cells. In the midst of this bright zone were small and large defects, imparting to the mucous membrane a fenestrated appearance even when seen with a magnifying glass. Portions of the mucous membrane were *extremely vascular*, but the greater portion of the vessels was thrombosed and obliterated; in some the contents were in granular disintegration, and wherever they were empty a *fatty* degeneration of the endothelial cells could be plainly demonstrated. In other places of the labyrinthian wall there existed a hemorrhagic infiltration of the mucous membrane.

Niche and Membrane of the Left Round Window.

The entire niche is hermetically filled by a large fibrinous clot, enclosing numerous red and some white blood corpuscles, an evidence of previous intense congestion. The laceration of the vessels of the destroyed mucous membrane of the posterior osseous frame of the niche could be demonstrated as the source of the hemorrhage; from this point pseudo-membranes could be traced into the centre of the

coagulated deposits. In the lower half of the membrane of the round window was a perforation, which was filled by coagulated fibrin, and was connected with a similar mass attached to the inner surface of the membrane in its entire surface of the scala tympani.

The niche of the right oval window and of the right stapes was filled partly by hypertrophied mucous membrane, partly by fibrinous exudations of the same character, as in the niche of the left round window; they were much larger, and enclosed larger groups of red blood-corpuscles, and more colloid bodies. Upon the promontory numerous lacerated blood-vessels could be seen. The limbs of the stapes were enveloped by the hypertrophied mucous membrane, which was covered by numerous granular cells. The stapedio-vestibular connection was normal. In the centre of the stapedial capitulum a portion of the bone was transformed into colloid substance (*cp.*, fig. 7); in that of the respective stapedial plate two such could be found (further mentioned later).

The histological changes in the labyrinth can be arranged *in two large classes.* The *first* class comprises changes due to a *purely mechanical condition*, coagulation of the lymph, gathering or aggregation of lymphoid cells, either isolated or in numerous groups occupying the *semicircular canals* and ampullæ, the inner cavities of the utriculus and sacculus, the right vestibule close to the free wall of the utriculus, and the cochlear turns of either petrous bone.

The results of some of these histological examinations are precisely the same as those in diphtheria, but they partially differ from these, inasmuch as the products of *retrogressive metamorphosis* of the lymphoid cells themselves and of the histological elements are perhaps less the *direct* than a *secondary consequence* of the morbific virus, owing to *thrombosis extending over the entire petrous bone.*

The *blood-vessels* of both petrous bones were tortuous and engorged. This globular stasis resulted in a general thrombosis, which extended through the entire labyrinth to the vas spirale and the capillaries of the stria vascularis. A few blood-vessels are empty. This general extension of the

thrombosis may be *explained by a more or less advanced fatty degeneration of the endothelium of the blood-vessels.*

As is well known, the blood remains fluid within the vessels as long as the epithelium is intact and the blood itself is in motion. The degenerated endothelia are probably partially detached, or perish by disintegration; and, since no regeneration takes place, the denuded rough places become the cause of coagulation and thrombosis. The thrombi *should, therefore, be regarded as autochthonic.* This condition of the endothelia corresponds closely with the observations first made by Ponfick that, in certain severe infectious diseases, the vascular endothelia undergo a fatty degeneration in great numbers, and are cast off. Ponfick[1] has demonstrated this condition in typhus recurrens in the living.

Special Condition of the Thrombi, according to their Situation.

Although the thromboses found in the blood-vessels, as stated above, may be attributed to a common cause, viz., the fatty degeneration of the endothelia of the vessels, their condition, nevertheless, presented a different appearance. In accordance with the character of the contents, *three large classes* may be distinguished.

The majority of the thrombi belong to the *first* class, comprising the smaller veins and the capillaries. Their contents consist mostly of red blood-corpuscles; the thrombus, nevertheless, does not present a red appearance; the red blood-corpuscles, being closely pressed together in consequence of the stasis, are irregular, flattened, and even polygonal. In an apparently later stage, they are markedly pale, in consequence of shrinking, defects are found, and a more or less wide and bright zone (*cp.* fig. 4) between the contents of the blood-cell and its wall. Besides there are found, mostly within the vessel, products of molecular detritus, and, if the products of disintegration are absorbed, smaller and larger defects as bright spots.

The *second* group is chiefly represented in the arterial and

[1] "Anatomische Studien über den Typhus recurrens."—*Virchow's Archiv*, vol. lx., p. 153.

venous vascular area of the *modiolus*. The thrombi are here plainly distinguished by a mostly *red*, sometimes orange, ochre-yellow, or clay-like *color*, although red thrombi are, as is well known, only met with in vessels of larger calibre. This alone should especially arrest our attention, the more so as G. Schwalbe[1] has recently studied and illustrated the anatomy of the vessels of the modiolus more thoroughly, and has also discussed them physiologically in a classical manner.

In the screw-shaped central canal of the modiolus an arterial coil is formed. Schwalbe gives an accurate description of the same in guinea-pigs, and distinguishes glomeruli arteriosi cochleæ majores, which, however, are not connected with each other, although their borders may overlap, and glomeruli arteriosi cochleæ minores. *According to Schwalbe, we have before us in this arrangement, as it were, a powerful current-breaker*, which causes an extraordinary diminution of the pressure and rapidity of the arterial blood, which enters through the cochlea and circulates within this beautiful apparatus, so well adapted to powerful resistances. This is effected (1) by the numberless windings, bendings, and loop-formations of the circulatory path, and (2) by the extraordinary elongation thus produced.[2] From the capillaries of the ligamentum spirale numerous veins develop, which run downward and thence as *radial* veins, with the upper side of the corresponding intermediary partition wall, centrally to the "*vena spiralis modioli*."

[1] Ein Beitrag zur Kenntniss der Circulationsverhältnisse in der Gehörschnecke (Contribution to the Knowledge of the Circulatory Relations of the Cochlea). *Cp.* Beiträge zur Physiologie (Contributions to Physiology), Dedicated to *Carl Ludwig*, on his seventieth birthday, by his pupils, p. 200–220. (*G. Schwalbe* gives a description of the anatomy of the ear in his "Anatomie der Sinnesorgane," 1887, p. 289–559.—*H. K.*)

[2] Although not strictly relevant, I cannot forbear quoting the following physiologically most important sentence, *l. c.*, p. 216: "If we review the circulatory conditions just described, there results a most surprising relation of the scalæ to the arterial and venous circulation. The scala tympani is surrounded only by *venous* vessels, while the *scala vestibuli* covers within its wall the *arterial vessels*. The scala tympani, which is separated from Corti's organ only by the thin membrana basilaris, escapes, therefore, the influence of arterial pulsations; all arterial paths surround the scali vestibuli, which itself is, moreover, separated from Corti's organ by Reisner's membrane and the entire ductus cochlearis. We may call the scala vestibuli the *scala arteriarum*, but the scala tympani the *scala venarum*.

It is obvious, that if these blood-vessels in the normal state already diminish by their tortuous structure the pressure and rapidity of the blood circulating in them, these factors must increase every pathological effect which produces stasis. The effect is, therefore, that in these vessels the signs of stasis appear earliest and most intensely. This is in accordance with the result of examination.

The red or yellow-colored thrombi within the tortuous blood-vessels of the modiolus consist of more or less numerous *parietal* leucocytes, with or without nuclei, which extend in places even up to the centre of the vessel, in the centre of the vessels of red, rarely normal, mostly already disintegrating blood-corpuscles, granular products of disintegration of the latter and of the blood-discs also, now and then white or gray irregular granular plates, the whole being interlaced with fine-threaded fibrin. In the thin transverse sections fine fibrin threads may be seen passing through the entire lumen of the vessel. In the centre of the vessel we do not unfrequently find, in consequence of complete disintegration and beginning absorption of the contents, empty spaces, as in the first groups. The orange-yellow or ochre-colored character of the contents is, of course, due to a diffusion of the blood pigment, and indicates probably an early development of the thrombosis. These thrombi resemble, with reference to the contents, mostly the "*leucocyte thrombus*," produced experimentally by Zahn.

From the thrombosed vessels of this second group an emigration of blood-corpuscles takes place into the perivascular lymph-space; for we find therein red blood-corpuscles, as well as cells containing blood-corpuscles. Schwalbe, in his above-mentioned work on the cochlea of the guinea-pig, describes a circumvascular lymph-space as well around the vena spiralis modioli as around the arterial wall of the glomeruli. I can confirm his statements in this respect with regard to man, and also with reference to his description of the existence of a fine reticular nucleated small band of connective tissue within the arterial circumvascular lymph cavity.

Thrombi similar to those in the modiolus were also found in the convoluted vessels, surrounded by bone, which send their branches to the maculæ and cristæ of the sacculi and ampullæ. The vascular wall, however, is here also not in direct contiguity to the osseous wall, but is separated from it by a perivascular lymph cavity.

The third group of thrombi is found in the *Haversian canals.* These are only exceptionally normal. But even then at least the form of the blood-corpuscles is no longer normal, but of the same character as in the first group. Others present, chiefly, besides products of disintegration of the blood-corpuscles, only *threaded fibrin;* after disintegration and absorption of the blood-cells the majority presents *only the threaded fibrin*, and consequently complete *obliteration* of the small canal.

Weigert has lately published a method of staining threaded fibrin with anilin upon the slide, a method which has afforded the most beautiful results in the changes of the Haversian canals just described.[1]

Sequelæ of Vascular Thrombosis.

a.—SEQUELÆ RESP. CHANGES IN THE VESSELS THEMSELVES.

If the blood-vessel remains thrombosed for a long period the nutrition of the vascular wall must of course be interfered with on the part of the vasa vasorum. We actually find in the different regions of the labyrinth, viz., in the modiolus and the perilymphatic space of the semicircular canals and ampullæ, a fatty degeneration or a complete destruction of the wall elements and consequently a bright, homogeneous wall, devoid of epithelium or a hyaline thickening of the wall, here and there with dark, fine streaks or curved lines as the last remnants of the cellular elements (*cp.* fig. 4.); the vascular wall even may in the area of the hyaline degeneration undergo such a complete disintegration as to leave only aggregations of the brownish disintegrated contents enclosed by fragments of the disintegrated vascular wall.

[1] *Cp.* On a New Method of Staining Fibrin and Microörganisms.—*Fortschritte der Medicin*, 1887, No. 8, p. 228 *ff.*

In the larger vessels, chiefly in the area of the arteria auditiva interna and its branches, the thrombosis leads to *necrosis of the vascular wall, to laceration, and to hemorrhages.* Such hemorrhages or blood-extravasations in the act of pigment metamorphosis were found in the trunk of both auditory nerves, also in the modiolus of the first cochlear turn (*cp.* fig. 1.); also laterally from the nervus ad sacculum and ad utriculum, and finally upon the mucous membrane of the labyrinthian wall and the niche of the round window. As far as the hemorrhages occur in the area of the acoustic nerve and its branches, the auditory nerves undergo, as already described in hemorrhagic pachymeningitis and in diphtheria, destruction and necrosis, and precisely as in the latter, accompanied by formation of granular cells, disintegration and atrophy of the nerves, and according to the size and volume of the hemorrhagic exudation we have smaller and larger defects in the nerve tracts. In the right acoustic such a defect measures $\frac{2}{5}$ *mm* in diameter. Similar but smaller defects were found on the side of the ramus sacculi (right petrous bone). We still see here and there in the defects the perineurium, deprived of its structure, occasionally studded with pigment granules in the shape of a rosary.

b.—INFLUENCE OF THE VASCURAR THROMBOSIS UPON THE INTERIOR MUSCLES OF THE EAR.

Unfortunately I have neglected to study the relations of the musculus tensor tympani in this direction, but I am able to offer information upon the influence of the thrombosis of the vessels of the stapedius muscle upon the latter and its nerves.

The Changes of the Stapedius—Partial Waxy Degeneration of the Muscle.

The respective specimens presented, even when seen with the naked eye, or with a magnifying glass, isolated bright spots or defects, reaching 1 *mm* in diameter, which only contained the empty sarcolemma tubes.

Staining with picrocarmine gave the muscular fibres, in which the transverse striation was still preserved, a beauti-

ful red color, while the altered fibres appeared greenish or green-yellow. The change begins in the centre of the muscle. Glycerine specimens, when examined with a higher power, showed that in some muscular fibres the nuclei and the transverse striation had disappeared, whilst the fibre itself appeared wider than normal, was colorless, homogeneous, gelatinous, or waxy, and, where the degenerated fibre protruded into the area of the above-mentioned defects, the gelatinous contents were already transformed into a finely granular detritus, and the fibre was collapsed and narrowed. Some fragments of the disintegrated muscular fibres were of a similar character. The sarcolemma was folded in places, otherwise normal. In the above-described defects we found an additional large number of normal sarcolemma tubes, covered in some places by groups of colloid bodies. The *nerve branches* supplying the stapedius muscle were partly in a state of disintegration of the myeline substance, partly in that of complete atrophy.

The blood-vessels in the interior of the muscle were thrombosed.

We have therefore before us a transformation of the contractile muscular substance of the primitive bundles of the stapedius, as was first described by Zenker as occurring in the larger muscles of the body after typhoid fever, in the form of waxy degeneration, and as was experimentally observed by Cohnheim [1] and Weil,[2] by the former after total ligature of the tongue, by the latter after ligature of its artery.

Finally, I will add, that the examination of the just-described specimens of the stapedius muscle gave a negative result with regard to microörganisms.

c.—INFLUENCE OF THROMBOSIS UPON THE LIGAMENTS AND THE ENDOTHELIAL CELLS OF THE SEMICIRCULAR CANALS AND THE AMPULLÆ, UPON THE SO-CALLED LABYRINTHIAN LIGAMENTS, UPON THE MEMBRANA PROPRIA, AND THE EPITHELIAL STRATUM OF THE MEMBRANOUS CANALS.

The fibrillar structure of the *ligaments* has disappeared; the fibrillæ are chiefly replaced by granules, arranged like a

[1] "Untersuchungen über die embolischen Processe."
[2] *Virchow's Archiv*, 1874, vol., lxv., p. 253.

string of pearls, which finally disintegrate; the ligaments thus become atrophied, rarefied, or perish. We therefore find in some specimens only a few narrow bands as remnants of the ligaments traversing the perilymphatic space. The membranous wall of the semicircular canals and of the ampullæ consequently loses its supports, it collapses and presents, instead of the sharp elliptical form, either the shape of a club, that of a pear, or that of a kidney, and in the extreme degree of atrophy of the ligaments the form of a horizontal paragraph mark, so as to bring the opposite wall in contact with each other. Besides, the *endothelial cells* undergo a retrogressive metamorphosis; in the earliest stage they show a dark, extremely granular appearance, or they resemble granular cells; this is also true of the lymphoid cells, which lose their nuclei prior to their complete disintegration. Finally all products of disintegration form a fine, molecular detritus, which is situated between the meshes of the remaining atrophied ligaments.

The membrana propria loses its striated character and presents a homogeneous, transparent appearance. The epithelial cells lose their nuclei, their framework frequently being alone preserved, or they disintegrate entirely. The canal is deprived of epithelium on its inner wall, exceptionally empty, or more or less filled by products of disintegration of the retrogressive metomorphosis, the material of which is furnished by the epithelial cells, as well as by the lymph of the endolymphatic space, or by the hyaline products (*cp.* fig. 3). The more or less complete filling of the endolymphatic space by such products of the retrogressive metamorphosis thus presents, in spite of atrophy of the ligaments, the collapse of the membranous wall (*cp.* fig. 3). As an accessory product of the tissue disintegration, a great many bundles of margarine needles are seen in the perilymphatic as well as in the endolymphatic space.

Results of Staining.

If we stain the above-described parts of the membranous hollow structures of the semicircular canals and ampullæ with picrocarmine, then *everything remains pale*, the mem-

brana propria becomes yellowish, and in no place does a red staining become apparent.

d.—INFLUENCE OF THROMBOSIS UPON THE PERIOSTEUM OF THE OSSEOUS SEMICIRCULAR CANALS AND THE AMPULLÆ.

The changes of the periosteum in measles differ from those in diphtheria essentially by the fact that in diphtheria the periosteum is either hyperplastic or atrophied, while in measles hyperplasia is never encountered. Moreover, the thrombosis produces throughout, in consequence of necrosis of the vessels, also a disintegration of the periosteum and a necrosis of the bone; its sharp outline is lost, and it appears sinuous, with jagged edges.

In those areas, in which a hyaline degeneration exists in the perilymphatic space, the periosteum may also take part in the hyaline transformation.

This *change of the periosteum* has a considerable *influence upon the nutrition of the adjoining osseous tissue*, producing smaller or larger defects with sinuous, ragged edges; these defects are sometimes filled by additional extremely fine and bright masses of detritus.

The osseous frame of these defects does no longer appear like connective tissue, but is bright, finely fibred, and poor in bone corpuscles. In the adjoining territories, which are free of defects, the bone corpuscles are no longer normal. Some are stunted, without processes, the majority in *fatty degeneration*, a change, apparently preceding the complete disintegration of the bone tissue, the products of which are sometimes, as stated above, encountered within the defects.

e.—INFLUENCE OF THE CHANGE OF THE HAVERSIAN CANALS UPON THE OSSEOUS TISSUE, VIZ.:

1. Influence of the obliteration;
2. Influence of the colloid degeneration of their endothelium upon the bone;
3. Influence upon the medullary substances.

1.—*Influence of the Obliterated Haversian Canals upon the Compact Bone Substance.*

The obliterated Haversian canals have throughout a finely striated character, and take with the above-mentioned *Weigert's* method of aniline staining a beautiful blue color upon the slide. If we examine a section from a territory of compact bone substance, stained with picrocarmine (fig. 5) *c. g.* from the cochlear capsule, we find in the midst of the bone tissue a larger or smaller number of unstained bright spots. These spots, resembling either narrow or wide homogeneous, gelatinous bands, are bounded by dark, mostly indented lines against the red-colored bone tissue (*cp.* fig. 5, lower fifth). These bright stripes apparently represent the basis-substance of the bone, in the act of decalcification or entirely decalcified. Occasionally one or more bone corpuscles are still seen in it, which have already lost their processes or present a spindle-like appearance. In many places the decalcified portions traverse the bone substance in a stellated manner, the latter like hazel-nuts bordering the already decalcified zones (fig. 5). We see in other places (*cp.* fig. 6) the further progress of decalcification, the bone tissue being in atrophy or disintegrating to a very fine detritus. In still other places this detritus, which is apparently extremely capable of absorption, is already absorbed. The bone appears fissured, multiple cavities develop, the bone substance is perforated like a sponge, and the different cavities are traversed by stripes or bands of the hyaline substance, appearing hyaline, which are moreover in places studded by the above-described detritus or very finely granular masses. Preserved portions of bone extend in places into the defects like islands, the bone corpuscles of which are already in the state of degeneration (*cp.* fig. 6).

2.—*Influence of the Colloid Degeneration of the Endothelium of the Haversian Canals upon the Compact Osseous Tissue* (*cp.* Fig. 7).

This mode of the osseous change was frequently found. In the earliest beginning we see a few endothelia on the

inner wall of the small canals transformed into round or oval, homogeneous, colloid bodies, and the remaining endothelia are in fatty degeneration. In an apparently later stage the greatest part of the calibre is filled by colloid bodies, and the blood-vessel, with its contents, is pushed aside; in the last stage the entire contents of the Haversian canals consist of extremely swollen colloid bodies, nothing can be seen of the blood-vessel or of the blood-corpuscles, the Haversian canal is already enlarged in the shape of a spindle, and finally colloid degeneration extends to the bone, where at its transition the Haversian canal is enlarged like a flask. But, of course, this process causes nutritive disorders, the effect of which manifests itself in the territories of bone adjoining the colloid masses by their presenting an appearance of finely striated connective tissue, in which the bone-corpuscles look like spindles, or are entirely absent.

3.—*Influence of the Obliteration of the Haversian Canals upon the Medullary Substance.*

The medullary substance is subjected either to a more or less complete necrosis,[1] in which the medullary cavity may be found entirely empty, or its contents undergo colloid metamorphosis. In the less intense degree of the process the colloid bodies lie between the meshes of the reticulum of the connective tissue; in the extreme degrees the entire medullary substance is in colloid degeneration. The mechanical and initiative influence upon the bone tissue bordering the medullary cavities may then be identical with those described sub. 2.

f.—INFLUENCE OF THROMBOSIS AND OBLITERATION OF THE HAVERSIAN CANAL UPON THE DEVELOPMENT OF THE OSSEOUS TISSUE OF THE COCHLEAR CAPSULE.

As is well known, the cochlear capsule ossifies by the mediation of cartilage. We find, however, even in adults,

[1] The necrosis of the medullary substance may have a double cause: the obliteration of Haversian canals, and the influence of a micro-organism (in reference to this see the bacteriological part).

still in the midst of compact osseous tissue, remnants of non-ossified cartilaginous territories. The obliteration of the Haversian canal was not without influence upon some of these cartilaginous territories.

In a specimen, stained with picrocarmine, 2½ *mm* medially from the mucous membrane of the promontory, an *unstained* zone, 1 *mm* long and ½ *mm* wide, was found in the cochlear capsule, consisting of cartilaginous cells in fatty degeneration, or already in a state of granular disintegration, the whole limited by normal cartilaginous tissue; the Haversian canals entering the borders of these bright spots were thrombosed. In another place, into whose boundary the likewise changed Haversian canals entered, the cartilaginous cells had undergone the *colloid metamorphosis*.

g.—INFLUENCE OF THROMBOSIS UPON CHANGES WHICH HAD EXISTED IN THE PETROUS BONE PRIOR TO THE OCCURRENCE OF MEASLES.

The above described changes in the middle ear, the hyperplasia of the mucous membrane, and the catarrhal affection of the middle ear have, I believe, preceded the measles. The thromboses of the vessels of the mucous membrane of the labyrinthian wall were brought about by the transformation of the round cells of the mucous membrane into numerous granular cells and colloid bodies, and by the partial fatty degeneration of the stroma of the connective tissue, in consequence of which the mucous membrane offered a macroscopically noticeable areolar appearance. The *complete absence* of epithelium in this region might be attributed to the thrombosis.

h.—INFLUENCE OF THROMBOSIS UPON THE NERVES.

Condition of the Nerves in the Maculæ and Cristæ.

A change of the nerves was noticeable where they ascend to the epithelium—*i. e.*, upon the connective-tissue stratum of the maculæ and cristæ. Also here a great number of the nerve fibres were still normal. The changed ones

presented either *a lustrous yellow, gelatinous*, or a *peculiar homogeneous, fatty appearance*, with preserved medullary sheath and apparently normal width, but with fatty degenerated nuclei of Schwann's sheath and atrophy of its protoplasm. This is probably the first stage of the atrophy. We have at any rate before us a *pure primary nerve atrophy, due to thrombosis.*

Condition of the Nerves between the Lamellæ of the Lamina Spiralis Ossea at their Point of Entrance into and beyond Corti's Organ.

Between the lamellæ of the lamina ossea, as well upon the surface as at the bottom, a disintegration of nerves takes place; in consequence of absorption of the products of disintegration an extensive formation of defects between the still preserved nerve fibres appears in places irregularly arranged (*cp.* fig. 8), or the nerve appears normal up to the neighborhood of the point of entrance. Hereupon the products of disintegration infiltrate the beginning of the membrana basilaris, and render its structure indiscernible (*cp.* fig 9 at 1'). Beyond the point of entrance the atrophy is usually complete. Within Corti's tunnel we can no longer distinguish an indication of the nerve fibre, and neither of the spiral nerve fibres; the fibres supplying the inner hair-cells are disintegrated immediately after entrance (*cp.* fig. 10, 11 *a*, and 13, 13). In no place are granular cells apparent. Since I succeeded in proving the occurrence of micro-organisms in the products of disintegration of the nerves between the lamellæ of the lamina ossea, I would like to explain the occurrence of necrosis of the nerves in this region as due to two factors, viz., to thrombosis and to a direct action of the micro-organisms upon the nerves.

Specially Noteworthy Results of Examination.

In the following I shall endeavor to describe specially noteworthy results of the histological examination. A repetition will be unavoidable where we are dealing in the different regions of the labyrinth with a recurrence of identical

changes in addition to those specially noticeable. As regards this, I ask the reader's indulgence in advance. For all noted changes cannot be comprehended from a general point of view, as, *e.g.*, in the discussion of the influence of thromboses upon the varied tissues of the petrous bones. Moreover, we do not present a mere enumeration of curiosities, for under those conditions the entire discussion would be valueless, but a description of conditions, which are either of general pathological interest, or, possibly, apt to give some explanation upon, as yet, unsettled normal histological points.

The Hyaline Degeneration.

Fig. 3 gives an approximate picture of this change, which is extremely variable and multiform. I will try in the following to give a complete description of the changes in the remaining specimens, but I am compelled to acknowledge that it will hardly be possible to give a full and elaborate description.

The endolymphatic space, except about $\frac{1}{4}$ to $\frac{1}{6}$ of its lumen, is densely filled up to the region of the epithelial layer by coagulated and disintegrated lymph, which no longer contains any lymphoid corpuscles. The epithelial stratum is indistinct. The last fourth to sixth of the lumen is filled toward the point of insertion of the membranous canal partly by a group of non-nucleated lymphoid bodies, partly traversed by a reticulum of hyaline fibres, which are studded toward the inner cavity by a number of hyaline spherical bodies, and thereby communicate with the filling mass.

The epithelial layer is in some places entirely absent, and, where it is still preserved, the epithelial cells are highly granular, or their framework only is visible. In other sections the epithelial layer is replaced by spherical bodies arranged like stalactites. The propria itself is mostly covered by hyaline bodies and molecular detritus, and, where it is visible, it is without fibrillar structure; in some places it is perforated: in these places the hyaline structures of the endo- and peri-lymphatic cavities communicate.

In the *perilymphatic space* we see neither ligaments nor their endothelial cells, but the entire space traversed, here by delicate, there by wider, hyaline reticula; in the meshes of the reticula lie here structures like granular cells or irregular clusters of molecular detritus and finely granular masses; there numerous gelatinous spherical bodies, or cactus- or coral-like masses, which are also hyaline in character, are situated upon the margin of the reticula. The *vessels* of the perilymphatic space are thrombosed, their contents are in many places in the act of disintegration. The cellular elements of the wall are devoid of nuclei or entirely absent, whereby the external wall appears homogeneous and transparent. The entire outer wall of the vessels may also have undergone hyaline degeneration, and assumes a rosary-like appearance from hyaline bodies, cut longitudinally and adherent to the wall. The external walls of adjoining vessels are sometimes connected by a chain of hyaline spherical bodies.

Whence is the Material of the Hyaline Degeneration Derived?

In the endolymphatic space the cells of the epithelial layer and the lymphoid corpuscles furnish the material for it. The hyaline mass may, to be sure, disintegrate again; this is proven by the connection of hyaline bodies not as yet disintegrated with the products of disintegration (*cp.* fig. 3: endolymphatic space).

In the perilymphatic space the periosteum, the membranous bands, their endothelial cells, nuclei of the stratum of connective tissue, and the lymphoid cells take part in the process. But this material alone would not suffice in order to fill the entire space with hyaline masses and reticula. We should rather assume that the hyaline degeneration has been preceded by a gathering, a conflux of lymphoid corpuscles, which have become polynucleated cells before the degeneration has taken place. This hypothesis is supported by the fact that in the portions undergoing hyaline degeneration groups of polynucleated cells are found (*cp.* fig. 3, z). All structures in the perilymphatic space furnish, together with collected lymphoid resp. polynucleated cells, the

material for the hyaline degeneration. *The entire condition is an excellent illustration of cell decay or coagulation necrosis* (*Weigert*).

Genesis of the Hyaline Degeneration.

The hyaline degeneration may be effected by the thrombosis or by a direct influence of micro-organisms or by both causes simultaneously. On account of the special relations of the micro-organisms in the territory of the hyaline degeneration, I am inclined to *ascribe the principal part in its development to the micro-organisms.* I have in no place observed their extraordinary spreading to such an extent as in the two sagittal semicircular canals, *i. e.*, in the endo- as well as in the peri-lymphatic space. They are here found as well in hyaline reticula as also upon the periosteum undergoing hyaline degeneration, along which they immigrate up to the adjoining bone; they are also found upon and within the hyaline bodies, and in quite unusual numbers in the molecular detritus. The latter condition is not surprising, since *Manfredi* also has proven in his experiments that, wherever the necrosis due to micro-organisms is mostly developed, *i. e.*, in the centre of the nodules, the micrococci are found in largest numbers, furthermore that the micro-organism is by no means destroyed by this necrosis. According to *Manfredi's* beautiful experiments (*l. c.*, p. 725), "it appears that the persistence of its vitality in the masses of the necrosed tissue is to be ascribed to its capacity to live even in the dilutest media, and in those most inappropriate for its nutrition."

Horizontal Semicircular Canal of the Right Petrous Bone.

Its *endolymphatic space* is filled by fine molecular lymph detritus, enclosing here and there non-nucleated lymph cells. The configuration of its membranous wall is no longer elliptical but pear-shaped. The epithelial stratum is disintegrated. The membrana propria can hardly be recognized, and is chiefly covered by molecular detritus.

The blood-vessels of the *perilymphatic space* are extremely tortuous and thrombosed, the contents partly shrunken,

partly disintegrated; the connecting bands either swollen or atrophied; the cellular elements in retrogressive metamorphosis; the periosteum proper partly atrophied, partly unravelled; the configuration of the adjoining bones either convex or concave. At the turn of the lateral into the medial limb, extending two *mm*, the *perilymphatic* space is altered in the following manner: From the wall of the membranous semicircular canal, opposite to the place of attachment of the canal, up to the periosteum of the osseous canal the space is filled *mostly by newly formed osseous tissue.*

Horizontal Ampulla of the Right Petrous Bone.

The blood-vessels near the point of entrance of the nerves into the crista, those of the crista itself as well as those of the perilymphatic space, are thrombosed; the wall elements of the latter are in fatty degeneration, their contents partially disintegrated; the membranous bands atrophied, their endothelial cells disintegrated. In the endolymphatic space several scattered groups of *non-nucleated cells, by which the cupula terminalis is covered throughout, and the space between this and the ampullar roof is completely filled.*

First Cochlear Turn of the Left Petrous Bone.
(*Cp.* fig. 1, pl. i. and ii.)

About the lower half of the first turn both scalæ are filled, the scala tympani completely, the scala vestibuli partly, by stratified masses, which are contiguous to the endostal periosteum of the cochlear capsule, but not blended with the latter; but they are throughout rather distinctly limited by the sharp outline of the periosteum from the osseous tissue of the cochlear capsule. The longitudinal diameter of the filling mass in the scala tympani measures about 2 *mm*; the interspace between the filling mass in the scala vestibuli toward the modiolus about $\frac{1}{2}$ *mm*; toward the ligamentum spirale about $\frac{1}{4}$ *mm*. The boundary of the filling mass in the scala vestibuli is formed toward the ligamentum spirale by Reisner's membrane. This membrane is pressed into the ductus cochlearis, which is thereby stunted.

Reisner's membrane itself is extremely thickened, its epithelium is undergoing fatty degeneration. Toward the cochlear axis the peripheral zone of the filling mass in the scala tympani which, stained with picrocarmine, remains bright, consists of non-nucleated and disintegrated lymphoid cells. The remaining stratified masses in both scalæ present already osseous transformation (*cp.* fig. 2), and were stained red with picrocarmine.

This examination resulted, as was intimated above, in an ossification of both scalæ of the first cochlear turn, *wherein the endosteal periosteum was not at all implicated*, in contradistinction to the condition found in a case of acquired deaf-mutism in the same region, which I have described, together with Steinbrügge in a former vol. of these ARCHIVES (*cp.* the respective illustration). In this case both scalæ were traversed by newly formed connective tissue and osseous trabeculæ, *issuing from the endosteal periosteum of the cochlear capsule.*

I consider the sharp limitation between the osseous tissue and the periosteum in similar results of autopsies as a criterion for deciding the question, whether we have to deal with a congenital or an acquired ossification in the cochlear scala. Politzer, *e. g.*, demonstrated at the eighth meeting of South German and Swiss aurists in Vienna, April, 1887, ten specimens taken from an unpublished case of ossification of the cochlea, which Burckhard Merian, in Bâle, had bequeathed to him. They were taken from a person thirty-six years old, admitted to an institution for deaf-mutes in his tenth year, but it was not known whether the disease was congenital or acquired. The cochlea, especially in the basal turn, was filled by osseous tissue, which was sharply differentiated in the specimen from the osseous cochlear capsule. According to this description I consider the condition as an acquired ossification, which may have developed from an ossification of gathered lymphoid cells in consequence of coagulation of lymph in the cochlear scalæ, produced by bacterial invasion after an infectious disease.

The changes in the territory of the cochlear canal are of a

double character. One series is *purely histological*, the other *purely mechanical*. The latter are the consequences of the former, for pathological processes resulting in atrophy or in formation of products of degeneration, produce a dislocation, a changed position either of entire parts of the organ or of their histological elements. Since in these dislocations the alteration of the ligamentum spirale takes a principal part, we shall place its description in the foreground.

The Changes of the Ligamentum Spirale.

(*Cp.* figs. 8 and 11, plates iii. and iv.)

In consequence of thrombosis of all its vessels the ligamentum spirale undergoes a nutritive disturbance of its histological elements, here to a slighter, there to a greater, extent. In the slighter degrees we have to deal with an equal atrophy of the cellular elements of all three layers, whereby the entire ligament assumes a brighter appearance. In the higher degree single layers, either the outer (fig. 11) or the middle, *disintegrate completely* with the formation of larger defects (*cp.* figs. 8 and 11) occasionally leaving behind products of disintegration, especially colloid bodies (fig. 8 at 3). The outer periosteal layer thereby loses, entirely or partially, its nuclei and its densely fibrous character, it becomes loosened or disintegrates completely, the adjoining bone becomes more like connective tissue or is transformed into osteoid tissue (*cp.* fig. 11). In the middle layer hyaline streaks are formed in places (fig. 11, h). The connection of the stria vascularis with the inner layer of the ligamentum spirale is loosened, or it is entirely detached (fig. 8), with the formation of a more or less wide defect. The epithelial cells[1] of the sulcus spiralis externus (fig. 8, 5 to 6) perish; they may be replaced by mono- or poly-nucleated cells (fig. 8, 5 to 6). This infiltration, as a rule, is in the region of the stria vascularis

[1] In very thin sections I found the epithelium of the ligamentum spirale situated above the prominentia spiralis still regularly arranged, but without nuclei.

wider, more abundant, and contains many cells, which have again lost their nuclei. In consequence of the described changes, the form of the entire ligamentum spirale is extremely altered. *Cp.* in reference to this the ligamentum spirale of fig. 8 with that of fig. 1.

Reisner's membrane (*cp.* fig. 8 and 9, plates iii. and iv.). The *histological alteration* of Reisner's membrane comprises all its elements; the membrane loses, by fatty degeneration, the endothelium of the vestibular surface as well as the epithelium of that facing the ductus cochlearis, and may finally appear structureless (fig. 9, 5). At the beginning of its origin on the vestibular side of the lamina spiralis ossea, and still some distance further upward and outward, the entire membrane shrinks by disintegration (fig. 8, at 9). In an entirely structureless condition the marginal portion may be covered by coagulated lymph or molecular products of disintegration (fig. 9, 6). Its surface is in other specimens covered by lymphoid cells (its outer two thirds in fig. 8, at 9), or by molecular detritus (inner third in fig. 8). There are sometimes on its margin hyaline bodies or clusters of either non-nucleated or nucleated lymphoid cells (fig. 8 in the middle third).

The *mechanical change* in Reisner's membrane is due to two factors: the first consists in the described dislodgment of the ligamentum spirale, its atrophy, and detachment of the stria vascularis from the underlying connective tissue; the second factor of the dislocation depends upon the atrophy of Reisner's membrane at its insertion upon the labium vestibulare of the lamina spiralis ossea.

The Changes of the Membrana Corti or Tectoria.

Everybody, even with a superficial knowledge of the topography of Corti's organ, inspecting the illustrations 8, 9, and 10, will be struck by the strikingly changed position of Corti's membrane. It is, as is well known, stretched above the sulcus spiralis internus, and extends to the outer hair cells beyond Corti's organ; it covers the latter, wherefrom the name "tectoria" is derived. In all

three illustrations no tectoria is really discernible; it lies at a great distance from Corti's organ above the sulc. spir. int. (fig. 9 and 10 or fig. 8), and is dislocated so far outward as to lie just above the crista spiralis.

We do not see its profile, as in other radial sections, but it is placed in such a manner as to present its entire lower surface (fig. 8, 13; fig. 9, 10; fig. 10, 8).

It is easy to discover that this *repeatedly* observed *condition* is not artificially produced.

The Changes of the Crista Spiralis.

If we view the figures 8, etc., we miss the concavity of the sulcus spiralis internus and the sharp-edged prominence of the labium vestibulare; the normally deep furrow is flattened, and the sharp-edged prominence of the labium vestibulare is represented by a blunt protuberance, apart from its infiltration with lymphoid cells. Embryological examinations prove (*Boettcher*, *Middendorp*, *cp. Schwalbe*, "Anatomy of the Ear," p. 356), that the acute angle of the labium vestibulare has as its precursor a slightly pronounced obtuse angle, which, however, assumes the definite form already during embryonic life. The illustrated morphological changes are possibly in connection with the tension and consequent detachment of the tectoria, which is intimately connected with the crista, and has been already discussed above.

The cells situated in the interdental sulci disintegrate, as also the epithelial cells upon the surface. The cells of the sulcus spiralis internus may be preserved; but they present a granular appearance (fig. 9 and 10), or they disintegrate partially (fig. 8 and 11); in this case they sometimes increase before disintegration and assume a darker shading, a granulation of the nucleus, which extends at the expense of the protoplasma of the cell-body close to the cell-wall (*cp.* fig 11). In this process of degeneration and disintegration also the labium tympanicum of the crista may take part, whereby it assumes a furrowed or fimbriated appearance (*cp.* fig. 10 and 11). The space of the sulcus spiralis

internus may at the same time remain free, or is more or less filled by lymphoid cells, or molecular products of disintegration (fig. 10). The latter are frequently superimposed upon the labium tympanicum, which thereby appears narrowed (fig. 8).

Changes of the Membrana Basilaris.

In vertical radial sections, as is well known, two strata of different structure are distinguished. The *upper* is a homogeneous continuation of the hyaline upper plate of the labium tympanicum ; it continues on the outer side into the hyaline lining of the sulcus spiralis internus. *Schwalbe* calls this layer the *membrana basilaris propria*. Upon the lower surface a tissue layer abundant in cells, the *tympanal lining stratum* (*Retzius*), is located as continuation of the lower lamella of the labium tympanicum.

The sharply pointed transition of the labium tympanicum into the basis substance of the membrana basilaris was lacking in several specimens. It can be distinctly recognized in fig. 9 and 11; it is not visible in fig. 8 and 11. in consequence of a deposition of products of disintegration of the adjoining nerve fibres. In some specimens the propria of the basilaris in its entire course up to the sulcus spiralis internus, where it continues as crista basilaris into the ligamentum spirale, is not visible on account of being infiltrated by lymphoid or polynucleated cells (*cp.* fig. 10). The same condition frequently existed in the nucleated hyaline layer situated upon the basilaris propria.

Mechanically, the outer portion of the basilar membrane is altered, inasmuch as in consequence of the atrophy of the adjoining ligamentum spirale it loses its tension, and instead of being horizontal, appears convex toward the vestibule. (*cp.* fig. 8 and 11).

The changes of Corti's organ are characterized by hyaline degeneration, partial or complete disintegration and atrophy of its cellular structures ; the nuclei remain longer preserved than the cell-body. A certain regularity in the succession of time of the disintegration can be distinctly recognized.

The inner epithelial cells perish earliest, followed by Corti's and Deiters' cells, then succeed Corti's arches, viz. the inner before the outer, and finally the lamina reticularis. The outer supporting cells resist longest, longer than Claudius' and Boettcher's cells. The various structures of Corti's organ still remaining, hereby frequently change their position, a dislodgment here in vertical, there in horizontal direction ; the nuclei are dislocated to places where they do not exist under normal conditions. The appearance of the atrophy in the different specimens are extremely multiform and varying, and its condition found in all specimens can hardly be described in all its details. A number of them is represented in fig. 8 to 11, and their exact description can be found in the explanation of the illustrations. It is, however, necessary to discuss the question, whether we have really to deal with atrophic changes or artificial products respecting the action of reagents.

"In reference to the rods," *Boettcher* says, "when it was still disputed, whether they were easily destructible or very resistible structures, to whose substance Henle ascribed even a hardness approaching that of cartilage, I have pointed out their difference in fresh specimens from those prepared with muriatic or chromic acid. They obtain their great resistibility from the action of these acids and can then be easier studied in certain respects." Since our specimens have been decalcified with chromic acid, the objection, that the illustrated *anomalies of the connection* of the rods are artificial and not pathological, loses its force. I believe upon the whole, that it would lay the facts under restraint, if we consider the described changes of Corti's organ as due to the action of decalcifying fluids. These can by no means produce a complete atrophy of the inner supporting and hair cells. I do not hesitate to put the described changes in relation to the thromboses in the ligamentum spirale, especially to the described alteration of the stria vascularis.

"We believe," *Schwalbe* says, "to recognize in the stria vascularis the organ which serves for the secretion of lymph." ("Organ of Hearing," p. 353). If this is, as in our case (*cp.* fig. 8 and 11), destroyed or degenerated, there

results a fundamental condition for the alteration of the lymph and for nutritive derangement in Corti's organ with its sequelæ.

Applications of the Results of the Examinations to Controversies upon the Normal Histological Structure of Corti's Organ.

Boettcher in his above-mentioned "retrospects," etc., has added to his own observation a full criticism upon the most important, still pending controversies upon the structure of the cochlea.

It is not within the province of my subject to enter into a discussion upon these. I may have another opportunity to make use of my specimens with especial reference to this. I shall at any rate point out to experts, that specimens presenting atrophic processes in Corti's organs are especially appropriate to decide certain points of controversy.

With reference to two points in dispute I concur entirely with Boettcher: first, that Corti's organ presents a third zone, the so-called *marginal cord* (*cp.* fig. 9 and 10) and furthermore, *that Corti's cells show a basal process.* This latter condition can undoubtedly be recognized in various specimens of mine. It is distinctly illustrated in fig. 9 and especially at *c* in fig. 11.

II.—Bacteriological Part.

General Literature and History.

E. Klebs[1] raises, in his "General Pathology," on p. 336, the following question: Are there any processes which can with sufficient reason be considered as produced by coccacea, and which, on account of their peculiarities, form a group of the same category?

If we answer this question, we should, according to Klebs, first consider the *acute exanthemata*, which have always been comprised in one group, on account of important affinities in the manner of their extension and

[1] General Pathology or the Study of the Causes and the Nature of the Processes of Disease." First Part: The Causes of Disease; General Pathological Etiology. Jena, 1887.

transmission, as well as in their clinical symptoms. After an explicit explanation of this sentence, he continues as follows: "This typical course, which manifests itself here more distinctly than in any other infectious disease, has brought to light the homogeneousness of the forms of disease, when nobody dared to dream of their parasitary nature. Now we may confidently form the postulate, that all these forms of disease *are produced by microörganisms of similar character.* They all have in common the epithelium as a suitable nutrient soil, and cause from this focus those local and general symptoms in cyclical succession. According to our present, although incomplete, experience, it cannot be doubted that they may be classed among the coccacea. This is sure in *variola*, doubtful in *scarlatina* and *measles*." Klebs has himself demonstrated that in variola the cocci show special arrangement, which he considers as an important characteristic, viz., the *arrangement in fours.*[1]

These *tetracocci* occur, besides isolated cocci, and those which lie together in irregular clusters, in the vaccine lymph as well as in the secretions of the mucous membranes of the pharynx and trachea in variola, and also in the vaccine lymph of man and cows. Cornil and Babés have also demonstrated the tetracocci in variola and in vaccine pustules.[2]

With regard to measles, full bacteriological examinations have been made of late by Cornil and Babés.

Cornil and Babés found in lungs affected with pneumonia, of patients who have died of measles, large numbers of diplococci, which, similar to the gonococci are composed, of two crescentic bodies, separated by a bright intermediary substance, form large zoöglœa masses or are arranged

[1] "In view of the wide distribution of tetracocci-forms, and especially in consideration of the fact that according to Bordone-Uffreduzzi, such forms are not missing among the microphytes found in the normal skin, the specific significance of the so-called tetracoccus variolæ (Klebs) seems to be quite doubtful," etc. "Text-book of Pathological Mycology," by P. Baumgarten. Second half, p. 389.

[2] With regard to the presence of micrococci in human variola, the first statements have been made, according to Klebs, by Luginbuhl, who considered the giant cells, demonstrated by him in the first beginning of the pustule, as bearers of the virus.

in chains. Babés' cultures with blood from the exanthema of measles, and also products of inflammation of the lung, pleura, lymph-glands (upon human and bovine blood serum), resulted in the formation of streptococci. Their members were 8-shaped micrococci, but some were also found resembling streptococcus pyogenes. Subcutaneously injected into the skin of the nose of young guinea-pigs, they cause redness of skin, fever, and sero-purulent conjunctivitis, changes produced in the same manner by direct transmission of the blood of a child affected with measles. (*Cp.* Klebs, *l. c.*)

L. Manfredi has published of late a lengthy paper on the bacterium of measles.[1]

In two cases of pneumonia following morbilli, the sputa were shown to contain Friedlaender's pneumonia cocci upon plate cultures. Manfredi found an additional coccus pathogenic to animals, which produced, in experiments made upon animals, a new typical disease, which induced the author to name the respect. microörganism "the micrococcus of the progressive lymphoma or granuloma in the animal body."

The shape of this microörganism is oblong, with rounded or obtuse ends. They are found chiefly single or as diplococci, sometimes more as bacillar elements. They stain readily without characteristic features. After subcutaneous injection, multiple granular neoplasms develop in form of nodules in the inner organs. In the resp. tumors the cocci are found most numerous in the centre of the cells, less frequently outside the cells, rarely in the blood-vessels; they have, therefore, a chiefly intracellular action. The lymph-vessels are the path of infection.

Own Observations. General Remarks.

From the former description of the changes found in the petrous bones, the fact of their almost complete accordance becomes apparent, a fact of great importance for the sub-

[1] Ueber einen neuen Mikrokokkus als pathogenes Agens bei infectiösen Tumoren. Seine Beziehungen zur Pneumonie. (On a new micrococcus as pathogenic agent in infectious tumors, and its relations to pneumonia.) *Fortschritte der Medicin*, 1886, No. 22.

sequent remarks. The readers, nevertheless, will be surprised, some even disappointed, that I did not succeed in proving the existence of microörganisms in a decisive manner in the *different* parts of the tissues, except in one of the two petrous bones first examined, *i. e.*, in the left. In the right I could demonstrate[1] the resp. microörganism only in the medullary cavities and in the products of disintegration of the nerves between the strata of the lamina ossea. It would be wrong, however, hence to conclude that it had been the principal factor of the changes only in the *left* bone. If some bacteriologists admit that the occurrence of microörganisms is sometimes not easily proven, even in experiments made on animals, this holds good the more for the resp. examinations on pathological specimens, and especially for those on the human petrous bone, which must be decalcified for weeks before it can be cut and the sections be bacteriologically examined. And then the investigator must be so fortunate as to succeed in making very thin sections, else the bacteriological examination is not practicable.

In addition, it is difficult to demonstrate the microörganisms in the tissues, especially in some territories of the labyrinth we have to deal with very small numbers of microörganisms, or the changes are old. We find, as is well known, microörganisms sometimes only in freshly diseased parts, *e. g.*, in erysipelas (Koch, Fehleisen), or the difficulty of their demonstration lies in the microörganism itself, the life of which may be only of short duration, it may degenerate quickly, etc.

Methods of Examination.

The examinations were made on sections, viz.:

(*a*) According to Manfredi's method, *l. c.*, p. 724, weak watery solutions of methylene blue. The sections remain in it many hours (it is not stated how many), are washed afterward in distilled water and treated with alcohol. Then clearing up in turpentine oil and preserving in balsam.

[1] In the two petrous bones of the second case, disfigured in consequence of too extreme decalcification, I succeeded in proving the existence of microörganisms, in the medullary cavities and in the membranous semicircular canals in hyaline degeneration of one side.

(*b*) The well-known *Gram's method*, also recommended by Manfredi, had furnished me with beautiful results, which were more distinct than those obtained with the former, because the sections were better decolorized. The resp. sections, preserved in xylol Canada balsam, furnished lasting specimens.[1]

(*c*) According to Gabbett's method of staining tubercle bacilli.

Gabbett's method of staining tubercle bacilli in slide specimens is a slight modification of that of Neelsen.[2] The staining solution, which, according to Gabbett, is superior to all other aniline staining fluids, is prepared as follows :

Solution I.—One part magenta red (English name of fuchsin), 100 parts of water, 5 per cent. carbolic water, 10 parts of absol. alcohol.

Solution II.—100 parts of a 25-per-cent. solution of sulphuric acid, 1–2 parts (grammes) methylene blue (the solution must be of a dark-blue color).

For the preparation of slide specimens solution I. is slightly warmed in a watch-glass until vapors begin to rise. The slides prepared as usual swim *two minutes* upon it.

The treatment with acids and the contrasting after coloring is now combined cleverly *in one act* by the use of solution II.

The slide is washed off quickly in water after use of solution I. and placed in solution II. for one minute. It remains only to wash in water, to dry event., and to preserve in Canada balsam in order to finish the specimen.

So far H. S. Gabbett.

Dr. Ernst gives the following results :

(1) Slide specimens, as well as sections, are beautifully stained in solution I. (for two minutes), and solution II. (for one minute).

[1] "The microscopical examination of the micrococcus in tissue sections is different in some respects, which, I believe, should be ascribed to two reasons : First, that this micrococcus retains the aniline colors only slightly after a certain period of its existence ; we can, therefore, after staining of the sections, not determine beforehand the means of decoloration for the tissue, and we run the risk of decolorizing also the parasites. Secondly, that the mentioned microörganism, which is rarely arranged in zoöglœa form, and, as a rule, rather attacks the protoplasm of the cells, whereby the nucleus is sacrificed by active segmentation and fragmentation, remains frequently exhausted in the midst of the not completely decolorized fragments of the nucleus."—Manfredi, *l. c.*

[2] *Cp.* according to Ernst, a review published in the *Medical Record*, July 9, 1887, concerning a note in No. 3,319 of the *Lancet*, April 9, 1887.

(2) It is unnecessary to *warm* the solution, a very desirable consideration for celloidin sections.

(3) Decolorization of the tissue and cells is complete to a degree never attained before, except by means of Futterer's modification (quite a tedious procedure); the contrast color thereby becomes much neater and cleaner.

(4) The staining fluids, as well as the colors in the Canada-balsam specimens (both in sections and in dry specimens), have remained unchanged in composition and intensity for three months.

(5) The single colored bacilli are brought out with great distinctness. The discovery of isolated bacilli, especially in the tissues, is extremely facilitated, probably because no red color whatever is left in the tissue.

From my own experience I can recommend this method as a very convenient and reliable one for sections of tissue containing cocci.

The form, arrangement, and size of the microörganisms, conditions and paths of invasion. (*Cp.* the colored plate.)

The microörganism in question should be ranked among the *coccacea*. It differs morphologically from that described by Manfredi by the constancy of the *round* form, *the spherical growth, while Manfredi describes his own as oblong, with rounded or obtuse ends;* the round form is constant, whether the parasite occurs as mono-, diplo-, and tetra-coccus (rarely[1]) in groups or irregular clusters, in chains or in strings. *The chain form* is, upon the whole, the *prevailing* one. The size of the single cocci varies, the larger ones being more numerous. If we place, morphologically, much value upon the chain formation, we should call this parasite also a streptococcus. *But it is very doubtful whether it may be considered as the specific microörganism of measles.* With reference to this point extensive investigations are necessary. I would like, however, to emphasize one point. In bacterial invasion of the labyrinth after diphtheria we have also to deal with a streptococcus and, as we know, with an *accidental and not with a specific microörganism* of diphtheria. But we know in this disease the conditions and paths of in-

[1] This rare occurrence as tetracoccus must be strongly emphasized with reference to the above-mentioned statement of *Klebs* concerning the tetracoccus in variola.

vasion, to wit, loss of the epithelium of the mucous membrane of the pharynx and the air passages, penetration into the lymph-paths, chiefly after development of tissue necrosis in the pharynx and then from the lymph-paths into the blood. If we now suppose that the streptococcus found in measles in the labyrinth is identical with that in diphtheria, therefore but *an accidental and not a specific microörganism*, the condition should be considered a complication of this infectious disease (of measles), like in various other diseases which are associated with affections of mucous membranes, as in variola, typhoid and puerperal fever. *Lesions* of mucous membranes, however, are in measles less frequent and less marked than in the just-mentioned infectious diseases; acute forms of pharyngitis and tonsillitis occur, to be sure, but, as far as I know, no necrotic destructions. Thus it only remains to assume an immigration of the streptococci into the lymph-path and thence into the blood, by means of a lesion of the epithelium of the mucous membrane developed from the catarrhal affection of the pharynx, tonsils, and air passages. The conditions of invasion are, at any rate in measles, more unfavorable for the streptococcus than in the other infectious diseases; the danger of a bacterial invasion of the labyrinth in this infectious disease is, therefore, much smaller. Affections of the labyrinth in measles, are, in fact, rarely (*cp.* the clinical part) observed. In my practice, extending over almost 30 years, I met with only the two cases described in the clinical part. The possibility of a more frequent occurrence, however, in autopsies, is by no means hereby excluded. If we consider that, as far as I know, the labyrinth of persons who died of measles had never before been carefully examined, and that my examination is the first of the kind, it would be a strange coincidence if I should find this extremely rare exception in the very first examination.

Regionary Occurrence.

The efforts to discover the microörganism in the different parts of the petrous bone proved frequently fruitless, especially in the right petrous bone, in which I succeeded in

finding it only in the medullary spaces and in the products of disintegration of the nerves, between the strata of the lamina spiralis ossea. On the other hand I found it in the left petrous bone, not only in this, but also in the Haversian canals, in the endo- and peri-lymphatic cavity of the semicircular canals, and especially in the region of the hyaline degeneration, and here again in large masses in among the hyaline products of disintegration. Furthermore, it was seen in the periosteum in hyaline degeneration, and along this up into the necrosed osseous parts described above, especially in their sinuses. I could not prove in the above-mentioned territories their intracellular occurrence, except in the medullary cells. I could count them here up to six, arranged in chain-form at the periphery of the medullary cell, near the cellular wall. They could also be demonstrated within the colloid spherical bodies of the medullary cavity. The results of examination in the cochlea were negative.

Biological Properties of the Microörganism.

After it has been found impossible to distinguish *morphologically* the microörganism found in measles from that observed in diphtheria in the labyrinth, we will try to prove this *biologically*. This consideration appears the more urgent inasmuch as there exist, as is well known, five different kinds of streptococci, which cannot morphologically be distinguished from each other, but the pathogenic actions of which nevertheless differ in many respects. If we now compare the respective histological results of examination of the labyrinth in diphtheria with those obtained in measles, we record, above all, the important fact that we found *in the former signs of new-formation*, viz., small-celled infiltration, *e. g.*, in the Haversian canals and the medullary cavities, and hyperplasia of the periosteum in different regions, *e. g.*, in the semicircular canals, in the periosteum of the pyramid of the petrous bone, *as well as necrosis*, *e. g.*, of the bone and the medullary substance, *while the examinations in measles demonstrated nowhere signs of new-formation, but only those of necrosis.*

Our results of examination agree with those of Manfredi with regard to this pathogenic action. He says (*l. c.*) that the formation of micrococcus used in his experiments appears to be a production of *cell-necrosis*, by destroying the nucleus *within the cell itself* and the structure of the cell. The proof of the penetration into the interior of the cells was brought, at least undoubtedly in the medullary cells, and the signs of necrosis in the different regions of the petrous bone were plainly visible. *The necrosis cannot, indeed, be considered everywhere a direct intracellular effect of the microörganism, but it is frequently only an indirect one*, produced by the thromboses in consequence of the mycotic fatty degeneration of the endothelium.

Paths of Degeneration in the Labyrinth.

The demonstration of the microörganism within the Haversian canals and the endolymphatic cavity proves the existence of the respective parasite within the paths of the lymph and blood. The manner in which the immigration thence into the labyrinth takes place may be learned from my investigations on bacterial invasion of the labyrinth after diphtheria, which explain the different possibilities on the basis of the special anatomical and histological relations of the labyrinth, particularly as regards the connection of the endolymphatic and perilymphatic spaces with those of the cranial cavity.

Brief Review of the Genesis of all Discovered Changes.

I consider in measles, as well as in diphtheria, the coagulation of the lymph a *purely mechanical condition*, due to the immigration of the microörganism. The disintegration of the lymph elements in the endolymphatic space, which is without blood-vessels, may depend upon an intracellular action of the parasite, and may then be considered as a mycotic necrosis. Their disintegration in all tissues containing blood-vessels may be effected by the same cause or may be the consequence of the thromboses, or both. The thromboses are the consequences of a mycotic fatty degeneration of the endothelium. Their consequences have already

been explained. I would like to point out the fact that from the thromboses the numerous noteworthy changes in the region of the ductus cochlearis can be explained. How much here, chiefly in Corti's organ, is due to a *direct* influence of the microörganism, cannot be stated on account of the negative results of examination.

The biological action of the parasite takes probably a prominent part in the production of hyaline degeneration, and also in the disintegration of the nerves between the strata of the lamina spiralis ossea.

The necrotic degeneration, or the complete atrophy of the medullary substance of bone is probably the consequence of the obliteration of the Haversian canals and of a *direct* influence of the microörganism upon its tissue. Each factor may, moreover, produce the same result.

The vascular necrosis also may have this double cause. It may develop, as is well known, in continued thrombosis, since it affects the nutrition of the vascular wall itself, but it may be also the direct consequence of the mycotic action.

III.—Clinical Part.

From the appended literature a comparatively great infrequency of affections of the labyrinth in consequence of measles becomes apparent. They seem to be still less frequent than those in consequence of diphtheria. I have observed only two typical cases, whose full report is justified by the rareness of such observations. They are characterized, like those after diphtheria, *by permanent complete deafness, by disturbances of equilibrium, viz., staggering gait.* For the explanation of these symptoms, which are fully discussed in my paper on diphtheria, the pathologico-histological results of examination, in the labyrinth of persons who died from measles furnish sufficient points of support. I do not enter here, therefore, into an additional discussion of these conditions.

HISTORIES OF CASES.

First Case. Complete Loss of Hearing with Staggering Gait in the Course of Measles.

I am indebted to the kindness of Dr. Habermehl in Edesheim, Palatinate, for the notes of this case.

"The girl, five years of age, was seized February 12, 1885, with measles and attended for three days. Besides the usual symptoms, fever, inflammation of the organs of the chest, extensive exanthema, etc., *hardness of hearing developed from the beginning, which soon resulted in complete deafness.* Hoping that the hearing would be restored with the expiration of the disease, I delayed active treatment until March 1st. I then made instillation of astringents into the external meatus, to which Dr. Stempel in Neustadt added vesicants. The epidemic was extensive, and had upon the whole a favorable character, with a few sequelæ. This child *alone* was afflicted with deafness, which doubtless has not improved since in the least, and seems to depend on a nervous paralysis, since paralytic symptoms were manifested in the peripheral parts, as was noticed in walking. The child stumbled in walking, moved rapidly onward without being able to restrain herself. Further observations could not be made since I made three visits, and the child was brought to me three times. This is the way in country practice."

The examination, which I made May 4th, revealed a perforation of the posterior half of the right drum-membrane, without a trace of pus, either in the external meatus or in the remainder of the drum-membrane or beyond the perforation (confirmed condition!). The left drum-membrane was drawn inward and presented considerable dulness of the mucous membrane. *Absolute loss of hearing for all sounds. Marked waddling gait.* After seven unsuccessful injections of pilocarpine the mother withdrew the child from treatment, which Dr. Habermehl was requested to continue at home. I have heard no more about the further course, but I suppose the deafness remained unchanged.

Second Case. Complete Loss of the Faculty of Hearing with Remaining Staggering Gait after Measles.

M. St., girl, twelve years of age, presented herself at the aural clinic for the first time May 30, 1885.

The mother states that the girl was five weeks previously seized with fever and intense headache, followed after a week by measles, from which recovery took place without particular complications. In the second week of the disease "the hearing was quickly lost, vertigo developed, and in the third week staggering gait when leaving the bed."

The examination revealed shortening of the cone of light on

either side and dulness of the mucous membrane of the drum-head. The patient is absolutely deaf and staggers still considerably. After twelve injections of pilocarpine, which proved entirely unsuccessful, the patient withdrew from treatment.

Review of Both Cases.

With the exception of the perforation of the right drum-membrane in the first case, which perhaps developed before the attack of measles, both cases present a complete agreement of symptoms—*absolute deafness and disturbance of equilibrium, viz., staggering gait.* Both cases can be explained by means of the above-described changes of the labyrinth, to the details of which I now merely refer. The *changes in the semicircular canals, or in the ampullæ* respectively, must probably be considered the anatomical causes of the disturbances of equilibrium. The deafness could be due to *hemorrhages* into the larger nerve bundles, to destruction of the nerves, and a great number of nerve fibres, furthermore to filling of the cochlear scalæ in consequence of stratified apposition of lymph cells with their consecutive changes, and finally to the fully described changes in the terminal apparatus, among which the colloid degeneration and the atrophy of Corti's organ rank first. I need hardly point out that no treatment can influence such changes, as its failure indicated in both cases. The number of my observations, however, is too small to conclude from them that the prognosis is unfavorable in all cases of nervous deafness after measles. They belong at all events to the severest forms of nervous diseases of the ear.

F. Rohrer's remarkable observation is a proof[1] that the prognosis need not always be absolutely unfavorable.

A girl, twelve years of age, was suddenly affected with complete deafness in both ears, a week after the eruption of measles. Beginning and course of the disease did not present any extraordinary features. Subjective noises on both sides, here and there with transition into distinct musical tones; staggering gait is not recorded. The examination of the history had to be made in writing on account of the complete deafness. Absolute loss of

[1] A case of sudden deafness after measles. *Correspondenzbl. f. Schweizer Ærzte*, vol. xiv., 1889.

hearing for all sounds, even for bone-conduction. Negative result of examination; slight swelling of Eustachian tube. Deafness lasting twenty-five days. Return of the faculty of hearing after headache of two days' duration, peculiar disturbances of vision, and psychical and nervous attacks; sulkiness, melancholia, fixed look, sluggish reaction of pupils, and a series of eclamptiform attacks.

Besides a central lesion, Rohrer says in the review of the remarkable case, there existed undoubtedly a certain connection of the affection with the measles and an affection of the tube and the middle ear. A diminution of the hearing power was probably thereby started. The subsequent absolute deafness, connected with profound disturbances of the nerve functions in general and of the acoustic nerves in particular, might have been produced by central changes and lesions. Hyperæmia of the labyrinth and a neuritic irritation of the acoustic from the middle ear may occur. Rohrer mentions here the immigration of coryza bacteria (Klebs) through the frontal and ethmoidal sinuses, and also through the tympanic cavity, whereby cerebral hyperæmia, especially of the base and œdematous swelling of the arachnoidal tissue and of isolated nerve sheaths may be produced. These serous infiltrations at the basis cerebri, Rohrer thinks, are under these circumstances easily produced; *transitory* severe meningeal symptoms, principally in children, may not infrequently be diagnosticated as consequence of these occurrences. One might at all events take exception to the hypothesis of the genesis of the complete deafness on account of hyperæmia and irritation of the acoustic, because these changes hardly produce absolute deafness. It may be perhaps more fully explained by the supposition of a hemorrhagic exudation at the acoustic, absorbed again in the course of weeks. The remarkable concomitant cerebral symptoms, which were associated with the return of the hearing-power, render, moreover, this case unprecedented.

Synoposis of the Literature on Measles as Cause of Aural Diseases.

1. I. Toynbee, "Diseases of the Ear," mentions—in chapter xv., under section 3—hardness of hearing produced by the action of

morbific poisons on the nervous apparatus of the ear, besides gout, typhoid fever, scarlatina, and mumps, also the measles.

2. On aural diseases as consequence and cause of general diseases. Inaugural Dissertation, by Joh. Heydloff, Halle, 1876. Measles in relation to scarlatina are considered as comparatively benign with regard to aural affections.

3. I. Gottstein. Contributions to aural affections developing in the course of acute exanthemata. *Arch. f. Ohrenheilk.*, vol. xvii., p. 16, etc. Among 1,193 aural patients treated by Gottstein in three years, 66 cases, or 5.5 per cent. were due to scarlatina, measles, and variola. G. describes here fully a case of acute desquamative inflammation of the drum-membrane in the course of measles, combined with perforative, purulent inflammation of the middle ear, recovery; and an additional case of throat diphtheria and *otitis diphtheritica* sinistra in the *second week of measles*, also with recovery.

4. L. Blau. On aural affections occurring in acute infectious diseases. *Deutsche, med. Wochenschr.*, 1881, No. 3. According to Blau, aural diseases, following measles, principally the purulent ones, may take a very severe course.

5. L. Blau. Diphtheritic inflammation of the external meatus after morbilli, followed after eleven days by diphtheritis faucium. *Berliner klin. Wochenschr.*, 1884, vol. xxi., p. 33.

6. Contributions to the statistics of aural diseases, by Dr. K. Bürkner. *Arch. f. Ohrenheilk.*, 1884, vol. xx., p. 81. We read on page 85: "Measles seem more seldom to cause aural affections. Of all patients, 3.0 per cent. ascribed their aural affections to morbilli, according to Kramer; 2.3 per cent. according to Schmalz; 2.7 per cent. according to Harrison; 1.5 per cent. according to Wilde; 0.1 resp. 0.4 according to Zaufal, and 3.2 per cent. according to *my* notes (epidemic of measles in Göttingen, winter 1881–1882)."

7. A. Tobeitz. Morbilli: clinical and pathologico-histological studies. *Arch. f. Kinderheilk.*, vol. viii., p. 321. Otitis media was the most frequent complication, 16 times among 73 convalescents. From the anatomical examinations of the hearing organ of the fatal cases there results, with all probability, a greater frequency of diseases of the ear. Of 22 children who died, the tympanic cavity was found, on examination, diseased in 17 cases. redness and swelling of the mucous membrane, destruction to the bone, obstruction with muco-purulent or putrid masses,

Clinical symptoms of disease were manifest in only 7 among these 17 cases. Tobeitz found in two, deceased on the first and third days, resp. after the appearance of the exanthema of measles, this otitis bilateralis also at the autopsy. Tobeitz supposes, therefore, that the middle ear, as well as the conjunctiva and the respiratory organs are *independently* affected in measles, but the affection may, clinically, not become evident before the stage of inflammation. The exudation of the, always lobular, pneumonia of measles is cellular, and tending to necrosis. The micrococci discovered in this affection are not concerned in the process of measles as such.

Literature Concerning Measles as an Etiological Factor of Acquired Deaf-Mutism.

1. "Diseases of the Ear," by I. M. Itard, German translation, 1822, p. 445, chapter xviii. On deafness by metastasis. Itard considers measles the disease which most frequently destroys the hearing by metastasis.

2. F. L. Meissner, "Deaf-Mutism and Instruction of Deaf-Mutes," Leipzig and Heidelberg, 1856, says, on page 158: "Extensive experience leads us to agree with Itard."

3. "Extension of Blindness, Deaf-Mutism, Idiocy, and Insanity in Bavaria," etc., by Dr. G. Mayr. No. 35 of the contributions to the statistics of the Kingdom of Bavaria, Munich, 1877. No. 42: In the year 1840 there were in Bavaria, among 2,897 deaf-mutes, 22, and in the year 1858, 3, in consequence of *variola and measles.*

4. A. Hartmann, "Deaf-mutism and Instruction of Deaf-Mutes," Stuttgart, 1880, mentions in table xii., p. 76, among 832 cases of acquired deaf-mutism, 30 in consequence of measles.

5. According to A. Hedinger, "The Deaf-Mutes and the Institutions for Deaf-Mutes in Württemberg and Baden," there was among 207 cases of acquired deaf-mutism in the two institutions of Baden, one case, or 0.48 per cent. after measles.

6. Schwabach. Deaf-mutism. S. Eulenburg's "Real-Encyclopædie." He mentions the rare occurrence of measles as cause of acquired deaf-mutism.

7. H. Schwartze, "Diseases of the Ear," Stuttgart, 1885, mentions, on p. 368, "larger blood extravasations into the labyrinthian cavity and into the membranous labyrinth," also in measles.

The statistics collected from the above-mentioned literature, except those of Tobeitz, indicate a comparative rareness of aural diseases in general after measles and of severe labyrinthian affections in particular. These statistical results offer a kind of consolation for the severity of the changes produced by the virus of measles in the labyrinth.

Explanation of the Illustrations.

Fig. 1 (Plates I. and II.).

Part of a radial section through the first cochlear turn and the neighboring region of the modiolus of the left petrous bone. That portion of the section is represented which lies nearest to the cochlear root. Hartnack, 22, tubus o.

We see in the modiolus portion, as far as possible to the left and below, two tortuous, dull and thickened fragments of the arter. auditor. intern. = g, partly limited by an upward concave line, an indication of a perineurium, which had become structureless = s p. Further upward a rust-colored hemorrhagic exudation in process of pigment metamorphosis.

g sp = ganglion spirale.
s t = scala tympani.
s v = scala vestibuli.
l sp = ligamentum spirale.
st v = stria vascularis.
v d c = stunted ductus cochlearis.

p = a bright, periosteal stratum of delicate fibre on the medial wall of the scala tympani with a vascular defect ; p′ = the same with two thrombosed vessels.

Both scalæ are filled, the scala tympani completely, the scala vestibuli partly, by a mass consisting mostly of lymphoid cells in osseous transition, the smallest portion of broken-down lymphoid cells without nuclei ; this area appears bright in specimens stained with picrocarmine, the remainder, which is in osseous transition, and which is represented by the portion designated by $\frac{3}{3}$ in fig. 2 (plates I. and II.) appears red.

The ossified masses are in contact with the periosteum of the cavities in question, but not blended with them. It is clear that a proliferation of the periosteum must be excluded. It is easy to discover that a filling of the scalæ was gradually brought about by a *stratiform apposition* of the cell masses. The sulcus spiralis

internus is filled by a mass, not morphologically discernible, and forms a connected and compressed whole with the papilla acustica, the membrana tectoria, and the region of Claudius' cells.

Fig. 3 (*Plates I. and II.*).

Transverse section through the membranous and osseous semicircular canal and the neighboring bone. Hartnack $\frac{3}{5}$, tubus o.

K = bone.
E = endolymphatic.
P = perilymphatic cavity.
h-h = membranous semicircular canal.
p = periosteum.
g = thrombosed vessels.
g^1 = thrombosed vessel with hyaline thickening of the wall.
z = group of cells with one and more nuclei.
P^1 = perilymphatic cavity with hyaline degeneration of reticular structure.

The outline of the osseous and membranous semicircular canal, which in the normal state is sharply defined as well as their elliptical form, is lost. The structure of the osseous frame of the semicircular canal is indistinct in parts, the periosteum partly unravelled, partly atrophic (*cp.* the periosteal lining at P^1). The endolymphatic cavity is almost entirely filled, chiefly by a homogeneous mass of detritus, with which (on the right of the drawing) a number of spherical hyaline structures are connected.

The epithelial layer of the membranous canal is lost; it is entirely lacking; the propria (in the upper half of the drawing) may only be in part plainly distinguished. The connective-tissue layer, the membranous bands and their endothelial cells, form with each other and with the mostly swollen periosteum, a coherent structure of a partly reticular, partly cactus- and grape-like hyaline mass. The blood-vessels are thrombosed at g^1 with marked hyaline thickening of the wall. At z a group of mono- and poly-nucleated cells is seen. The region indicated by two lines $<$ is represented in the following figure with higher power.

Fig. 4 (*Plates I. and II.*).

Hartnack $\frac{8}{5}$, tubus o.

In this enlarged representation the grape-like form of the hyaline degeneration is plainer, the condition of the hyaline masses, which are again partly molecularly disintegrated, more coarsely

granular on the left and below the blood-vessel, which is traversed by the section, and whose wall is considerably thickened, more delicately granular below the longitudinal section of the blood-vessel.

In the latter is specially noteworthy (1) the condition of the *wall*. Its bright appearance is interrupted by dark long and short lines, apparently still the indications of the cellular elements in hyaline transition. And (2) the contents. The blood-corpuscles are irregular and mostly polygonal in consequence of the formerly existing globar stasis.

Fig. 5 (*Plates I. and II.*).

Part of a *vertical section* through the right cochlear capsule. The drawing is taken from a spot situated 1 *mm* medially from the mucous membrane of the promontory. Hartnack $\frac{3}{5}$, tubus o. Specimen stained with picrocarmine.

The specimens represent the initial and final stage of the change in the bone due to obliteration of the Haversian canals. The initial stage is represented in the lower $\frac{1}{5}$ of the drawing. The irregular appearance of the bright spots in the lower $\frac{1}{5}$ is caused by the basis-substance in the act of decalcification, which remained unstained by picrocarmine. The osseous areas lined by them, in which the bone corpuscles have partly lost their processes, were stained pale-red with picrocarmine. The upper $\frac{4}{5}$ represent, as far as they are characterized in the centre by large lacunæ, the final stage of the bony change due to obliteration of the Haversian canals. One part of the basis-substance of bone is still preserved in form of bridges and strips, which are covered in part by products of disintegration ; another abounds in the shape of arch-like lines, which are connected with each other by small and large bright spaces.

These are complete osseous lacunæ, originating from the breaking down of the osseous tissue into detritus, with subsequent absorption. The Haversian canals (on the left of the illustration) contain only thready fibrin, and present a streaked appearance ; another canal (on the lower right) shows shrinking of the contents besides the striated appearance.

The osseous tissue, situated on the right and left from the above-described upper $\frac{4}{5}$, shows pale-red coloration, finely fibred appearance, partly with stunted and partly without any bone corpuscles ; the same holds good for the immediate neigh-

borhood of the obliterated Haversian canals. The Haversian canals on the left remained colorless, but every thing else showed yellow picrin staining, as represented in the illustration.

Fig. 6 (Plates I. and II.).

Part of a *horizontal section* through the right cochlear capsule. The drawing is taken from a place in the bone, which is situated 2 *mm* laterally from the inner cavity of the second cochlear turn. Hartnack $\frac{8}{9}$, tubus No. 0. Glycerine specimen.

The changes represented in the drawing are, on the whole, the same as those presented above as the final stage of the upper $\frac{1}{5}$ in fig. 5, on the longitudinal section, only differing in their being extended over a larger territory. Besides the numerous interspaces due to the destruction of the bone tissue, there is seen in the centre of the drawing a still preserved oval island of bone, with bone corpuscles without processes, surrounded partly by decalcified basis-substance, partly (on the left and below the bone-island) by an interspace. Both Haversian canals (on the right of the illustration) are thrombosed. The preserved osseous stroma is chiefly covered by a finely grained, homogeneous detritus, or the meshes produced by it enclose these fine masses of detritus.

Fig. 7 (Plates III. and IV.).

From a transverse section of the capitulum stapedis of the right temporal bone. Hartnack $\frac{8}{9}$, tube drawn out.

H K = Haversian canals.

bk and bk′ = remnants of parietal blood corpuscles.

As far as H K extends the calibre of the Haversian canals is unchanged ; in consequence of the stasis, the blood-corpuscles are mostly polygonal. The greatest part of the Haversian canals is considerably enlarged by partly roundish, partly oval colloid bodies. Remnants of blood-corpuscles are seen only at isolated points of the space, whose walls are enlarged and from which the epithelium is absent. The epithelium also is wanting throughout the whole length of the lower wall, between bk and bk′.

Fig. 8 (Plates III. and IV.).

From a radial section through the second cochlear turn and the adjoining cochlear capsule. Hartnack $\frac{8}{9}$, tubus 0.

1. Osseous cochlear capsule. 2. External or periosteal layer of the ligam. spirale. 3. Region of the middle or loose layer

(atrophied). 4. Inner layer, g = thrombosed vessels. 5. Transsparent substance of the crista basilaris, which, at 6, goes almost at right angles into the membr. basilaris instead of a concave curvature, as in the normal state, while the mb. is curved instead of horizontal. Claudius' and Boettcher's cells are missing in this region. In their place products of disintegration are seen. Between 5 and 6 the cells of the sulcus spiralis externus are also wanting. Instead of these mono- and poly-nucleated cells are seen. 8. Region of the stria vascularis. 9. Reisner's membrane. 9′. Its insertion to the detached ligam. spirale. 9″. Reisner's membrane atrophied at its insertion. 10. The radial and medullated nerve-fibres between the layers of the lamina spiralis ossea. The numerous regularly recurring light spaces are due to lost nerve-fibres. Cr. = Crista spiralis. 11. Its labium vestibulare is infiltrated with lymphoid cells. This infiltration continues to 9″ and 15, beyond the margin of the crista spiralis. 12. Labium tympanicum of the crista. This presents chiefly a homogeneous appearance, in smaller portions a striated one, except at its inner portion, where (to the right of 9″) on the upper surface of the nerve extension a few spindle-like, radially arranged nuclei of the periosteal layer of the crista may be seen. 13. Membr. Corti, or tectoria, is visible *en face* and dislocated. The tectoria is chiefly blended with Reisner's membrane ; the blended portions are concavo-convex. 14. Marginal cord of the tectoria covered by mono- and poly-nucleated cells and products of detritus. 15. Its inner fixed insertion in the act of disintegration. 16. Sulcus spiralis internus ; its epithelium partly in the act of disintegration, partly wanting. The transparent substance of the labium tympanicum of the crista (12) in the act of disintegration, narrowed, and its surface furrowed, instead of being smooth ; under the surface products of disintegration, partially covering the initial portion of the basilaris, hence its transition into the latter is indistinct. The nucleated layer of the basilaris devoid of nuclei, the initial portion of the latter is striated. The tympanal lining stratum (*th.*) of the basilaris apparently widened. This is due to an infiltration of non-nuclear as well as mono- and poly-nucleated cells, which extends into the area of the nerve on its lower surface (near 12). 17. Inner, 18. Outer rods of Corti's arches. 19. Outer supporting cells.

With regard to the characteristic change of the ligamentum spirale, we are best instructed by the large crescentic, almost

entirely empty space (3), in the lower portion of which a large colloid body is situated, which has been forced arch-like into the corresponding portion of the ligamentum spirale (in the normal state it is concave towards the scala vestibuli). The falciform space represents the so-called *middle loosened layer* (3) of the ligamentum spirale (Schwalbe) perished by tissue disintegration, whose ultimate product is the hyaline body. The *outer*, in the normal state, dense, periosteal layer (3) is preserved, but loosened and has mostly lost its former abundance of nuclei. The adjoining bone is also changed (*cp.*, the text). The right lower five oval spaces are veins with shrunken contents. The left thrombosed vessel, situated to the left and somewhat below the colloid body, is a vein. Downward and inward from this we see an atrophic portion, which, as well as the thrombosed vein, *belongs to the inner and, normally, dense layer* (4) *of the ligamentum spirale*. This third wide layer of the ligamentum spirale, which normally consists of densified connective tissue, abundant in cells, has a brighter appearance than in the normal condition, on account of one portion of the still preserved cells being devoid of nuclei, another being entirely destroyed. The extreme stage of atrophy is found below the colloid body, upon its inner and lower portion (*cp.* the text above).

Upon the inner surface of the inner layer of the ligamentum spirale, facing the sulcus spiralis externus, upward from the insertion of the basilar membrane, we see the translucent limiting membrane (5) in connection with the basilaris, forming its modified continuation. It is studded with a number of mono- and poly-nucleated lymphoid cells, which have partly lost their nuclei. Their epithelium is wanting; it is lost. Where it ends the considerably changed stria vascularis (5) begins. This, in the normal state intimately connected with the inner layer of the ligamentum spirale, has become detached from it by the pathological process; the point of detachment is indicated by an arch-like interspace, expanding upward to the insertion of Reisner's membrane (*g*[1]). Reisner's membrane is hereby already lowered at its insertion and its angle of insertion, outer-upper or vestibular angle, becomes obtuse, while in the normal state this does not occur below the third turn. Partly in consequence of this detachment, partly through the morphological change, the shape of the ligamentum spirale in the entire region of the scala vestibuli is changed beyond recognition. In the normal state[1] concave from the

[1] *Cp.* Fig. 1.

insertion of Reisner's membrane to the prominentia spiralis, on this place convex, further downward again concave, and throughout with a sharp edge, the big difference is well marked, notably by the absence of the prominentia spiralis and its vessel. The blood-vessels of the ligament are obliterated. Instead of the ligament we see an irregular layer of mono- and poly-nucleated cells, which are devoid of nuclei.

Corti's organ is considerably changed. Upon the membrana reticularis, to the end of the outer supporting cells, we see, partly, a number of hyaline bodies whose walls are already in the act of granular disintegration, partly fine detritus and coagulated lymph. The inner supporting (the hair) and the epithelial cells, situated inward and below these, are wanting. Instead of these we see detritus, a few mono- and poly-nucleated cells, the largest ones being outward from the body of the inner rod.

The nerve-fibres passing in- and outward from the inner rod of Corti's arch to Corti's organ, are absent. The configuration of Corti's tunnel (between 17 and 18) is irregular, in consequence of the altered curvature of the arch, especially the inner one. The basal cell of the outer rod, as well as the protoplasmatic layer, are wanting.

Specially noteworthy is the change in the region of the outer supporting cells. The membrana reticularis extends only to the fourth last outer supporting cell. The second last is already atrophied ; its dislodged nucleus lies next to the third last. The limiting walls also of the the four last supporting cells are already in the act of atrophy, and their fine products of disintegration are confluent with those of Claudius' cells. The basal portions of Deiters' cells are partly in hyaline degeneration, partly atrophied. Of their phalangeal processes the process of the second and fourth cells is the most plainly visible. The atrophy of the hair-cells is further advanced than that of Deiters' cells. We see the remnants of the first and fourth hair-cell adhering as hyaline bodies to the membrana reticularis.

Fig. 9 (*Plates V. and VI.*).

From a radial section of the second cochlear turn of the right petrous bone. Hartnack $\frac{3}{8}$, tubus drawn out.

1. Radial medullary nerve-fibres. 2. Crista spiralis. Its surface, instead of being slightly convex, is irregularly convexo-concave, studded with mono- and poly-nucleated cells. This in-

filtration continues into the sulcus spiralis internus. The transparent layer, the vitreus of the crista, is dull. The processes of the stellate bodies of the propria are absent. The propria and the periosteal layer form a stratum, interlaced by radially arranged nuclei, upon whose surface up to the neighborhood of the labium vestibulare (3) lies a cylindrical epithelium, the outlines of which upon the inner half of the row of cells are irregular and indistinct on account of beginning disintegration. 3. Its labium vestibulare is stunted (*cp.* text). 4. Its labium tympanicum, between both the sulcus spiralis internus, is flattened, instead of being concave. 5. Reisner's membrane. 6. Transition of the labium tympanicum into the basilar membrane. 7. Nuclear layer of the basilar membrane. 8. The so-called tympanal lining stratum infiltrated by mono- and poly-nucleated cells. 9. Epithelium of the sulcus spiralis internus, partly extremely granular. 10. Corti's membrane, with its marginal cord. 11. Inner; 12. Outer Corti's rod. 13. Outer supporting cells. 14. Remnants of atrophied Corti's cells. 15. Atrophied basal pieces of Deiters' cells, compressed by a transversely oval, hyaline structure. 16. The epithelium of Reisner's membrane (5) is wanting; it is mostly covered by products of retrogressive metamorphosis; between its inner portion, which is here abnormally convexo-concave, and the centre of Corti's membrane, or m. tectoria (10), coagulated lymph is found; the tectoria is detached from Corti's organ and placed upon its surface. *All structures of Corti's organ, situated in front of the inner Corti's rods, are completely destroyed, viz., the inner supporting cells, also the nerves running to the inner auditory cells, the inner auditory cells themselves, and the epithelial cells* (*Retzius*), *situated inward and below the auditory cells.* Instead of these we see between 4 and 11 a group of polynucleated cells, lying outward and somewhat upward from 11 upon the inner arch, two oval, hyaline structures (possibly products of degeneration of the inner hair cells), the latter surrounded partly by some polynucleated cells, partly by coagulated lymph, which covers and encloses the entire remaining Corti's organ.

Of both Corti's arches, only the outer one shows fibrillar structure, the inner only upon the upper portion, the basal portion being homogeneous. The outer basilar portion is lacking. We see Corti's tunnel, filled by an oval structure, and enclosed by an anchor-like, dark, homogeneous mass, partly contiguous to the inner rod, and by a bright, hyaline mass, which embraces the

inner surface of the outer rods. Of the phalangeal processes of Deiters' cells, we see only two distinctly, viz., the first and the last, but all their basal portions, stunted, and approximated by a large, transverse, oval structure, situated between the last Deiters' and the first outer supporting cell.

We see three stunted hair-cells without hairs, two of which, the two first, have distinct processes directed towards the basilaris.

The structure of the outer supporting cells still appears normal, except the indented condition of their boundaries, directed toward the ductus cochlearis, which in the normal state is convex.

Fig. 10 (*Plates V. and VI.*).

An additional radial section from the second cochlear turn of the right petrous bone. Hartnack $\frac{8}{9}$, tubus drawn out.

1. Radial medullary nerve-fibres at 1′ disintegrated. 2. Crista spiralis, almost entirely covered, *i. e.*, the inner half by nucleated, and its outer half by cells without nuclei, and by products of disintegration. 3. Its stunted labiumve stibulare. 4. Its labium tympanicum. The transition into the basilar membrane hidden by overlying products of disintegration of the nerve and invisible. The surface of the labium tympanicum is partially disintegrated, thereby presenting a fimbriated appearance. 5. Nuclear layer of the basilaris partially covered by products of disintegration. 6. Basilaris propria, only its outer half visible. 7. The so-called tympanal lining stratum infiltrated by mono- and poly-nucleated cells. The infiltration partly extends into the membrana basilaris, which, therefore, appears indistinct. 8. Corti's membrane with its marginal cord. 9. The flattened sulcus spiralis internus with its epithelium, upon which products of disintegration are situated. 10. Region of the inner rod. 11. Outer rod. 12. Outer supporting cells. 13. Disintegrated nerve running to the inner portion of Corti's organ. The structures of Corti's organ, situated in front of the inner rod, are entirely missing, as in fig. 9. Instead of the inner rod (10) we see a pear-shaped hyaline structure partly covered by an oval, dark, nucleated cell, the nucleus itself being highly granular. The body of the inner rod is missing; only its head-piece is seen. In the corresponding interspace we see a large and next to it a smaller hyaline structure, both surrounded below by an isolated cell, above by a group of polynucleated cells, the entire territory in question being outwardly limited by molecular detritus.

The outer rod (11) has lost its delicate, dark, longitudinal striæ partly upon the basilar portion, its head-piece, as well as that of the inner rod, being more striated than in the normal state. The protoplasmatic lining and the basilar cells of both rods are absent. Instead of these we see Corti's tunnel chiefly filled by a round and hat-like hyaline structure, the latter with two nuclei. The cell upon the outer rod (11) is perhaps the outer basilar cell, which has advanced further upward. The entire condition probably represents a product of metamorphosis of the basilar cells and of the protoplasmatic lining of the rods. The group of cells between 4 and 13 is a remnant of the perished portion of Corti's organ, situated in front of the inner rod. Nerves are not seen to enter Corti's tunnel. Three stunted hair-cells (14) are seen, all without processes, and just as many changed Deiters' cells.

Of the Deiters' cells (15) the first is blended with the first hair-cell, both are stunted, the last, the fourth Deiters' cell being the least stunted. It represents a concavo-convex arch, apparently interrupted by an overlying cellular structure, which can be traced to the neighborhood of the basis. The basal portion of the first Deiters' cell is in hyaline degeneration, between which and the basal end of the last an irregular dull granular mass is situated. The Claudius' and Boettcher's cell are absent. The entire Corti's organ is enclosed partly by coagulated lymph, partly by molecular detritus.

Fig. 11 (*Plates V. and VI.*).

Radial sections through the second cochlear turn of the *left* petrous bone. Hartnack $\frac{3}{4}$, tubus O.

1. Bone. 2. Inner or periosteal stratum of the lig. spirale, loosened at 2′, inwardly a hyaline band; downward from 2 at 2″ we still see the nuclei of the periosteal layer, in the remaining portions to the parts below more or less large defects, due to tissue disintegration. This is also true of the middle and inner layers in the upper portion, and of the middle layer in the lower portion of the drawing. g = thrombosed vessels. 3. Transparent substance, continuation of the propria of the basilaris. 4. Region of the prominentia spiralis in the act of atrophy and disintegration with a thrombosed vessel, downward a group of lymphoid cells, which further downward are lying upon the transparent substance (3) of the basilaris. In the direction of the outer supporting cells the basilaris shows indications of the disintegrated Claudius'

cells. 5. The tympanal lining stratum, infiltrated by lymphoid cells, up to the radial medullary nerve-fibres. The layer which lies nearer the labium tympanicum is brighter, and in the act of atrophy. 6. Radial medullary nerve-fibres, their entrance into the epithelium (*a*) atrophic; beyond their entrance, granular products of disintegration. 7. Labium tympanicum of the crista spiralis. 8. Its transition into the basilaris. 9. Region of the sulcus spiralis internus; the inwardly situated epithelial cells partly spherical, in beginning disintegration, instead of low, as in the outward portion. 10. Region of the inner supporting and hair-cells, which are lacking. In their place upward some detritus. 11. Inner Corti's arch defective. 12. Outer Corti's arch. Its body proper separated from the head and basilar portion, and somewhat dislocated with the separated ends. Basilar cells and protoplasmatic layer of both rods absent. 13. Nuclear layer of the basilaris, covered by products of disintegration; these products partly cover the adjoining propria of the basilaris. 14. Outer supporting cells, their outer wall partly covered by polynucleated cells. The protoplasma of the four outer supporting cells is homogeneous, and brighter than that of the remaining portion. The greatest portion of Corti's and Deiters' cells is atrophied. Of Deiters' cells, the last (*d*) is preserved best. *We emphasize that the process of one Corti's cell*, the cellular body of which is in hyaline degeneration, *is still preserved running towards the basilaris.* The inner portion of the membrana reticularis is absent.

Explanation of the Colored Plate (*Tab. VII.*).

Fig. A.—From the perilymphatic space of the sagittal membranous semicircular canal of the left petrous bone. From a visual field of the hyaline degeneration, with partial disintegration of the hyaline masses. *Hartnack.* Oil immersion $\frac{3}{12}$. Tubus 0, 650:1. We see chiefly mono- and strepto-cocci, now and then also irregularly arranged groups and clusters of cocci.

Fig. B.—From a medullary cavity of the left petrous bone. *Hartnack.* Oil immersion $\frac{4}{12}$. Tubus 0, 1000:1. We see chiefly mono- and diplo-cocci, isolated chains, and clusters of cocci. The medullary substance was partially destroyed, therefore a portion of the medullary cavity empty.

Fig. C.—From a Haversian canal of the left petrous bone. *Hartnack.* Oil immersion $\frac{4}{12}$. Tubus 0, 1000 : 1. We see mono- and isolated tetra-cocci; but the chain-form is, upon the whole, prevalent.

Fig. D.—From a Haversian canal of the right petrous bone. *Hartnack.* Oil immersion $\frac{4}{12}$. Tubus 0, 1000 : 1. All forms mentioned before are also represented, but in smaller number.

Fig. 1.

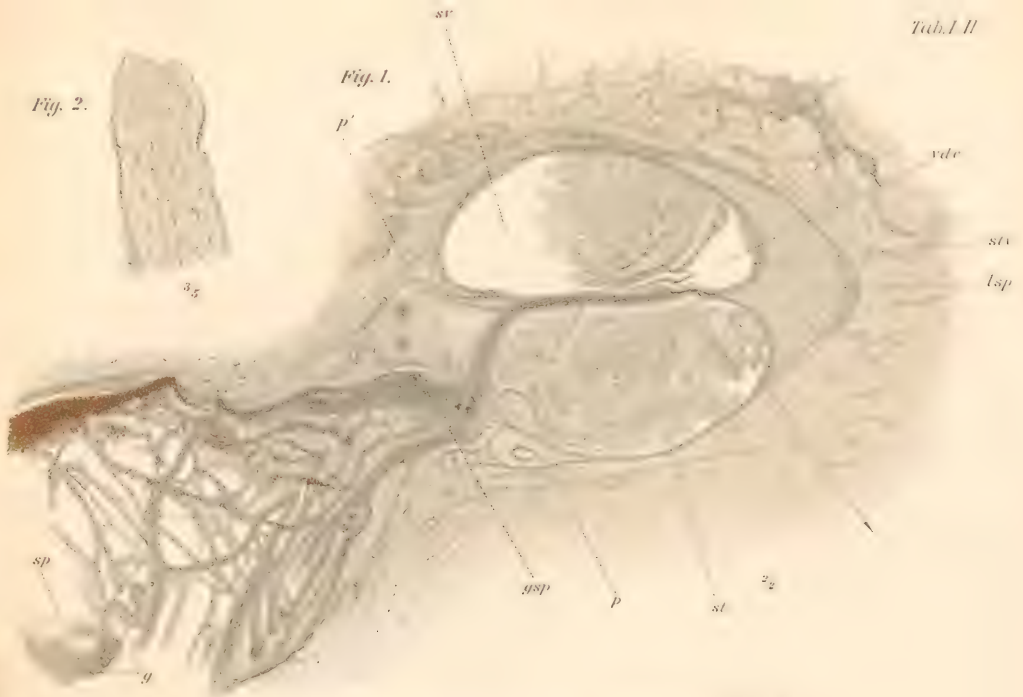


Fig. 2.

Fig. 3.

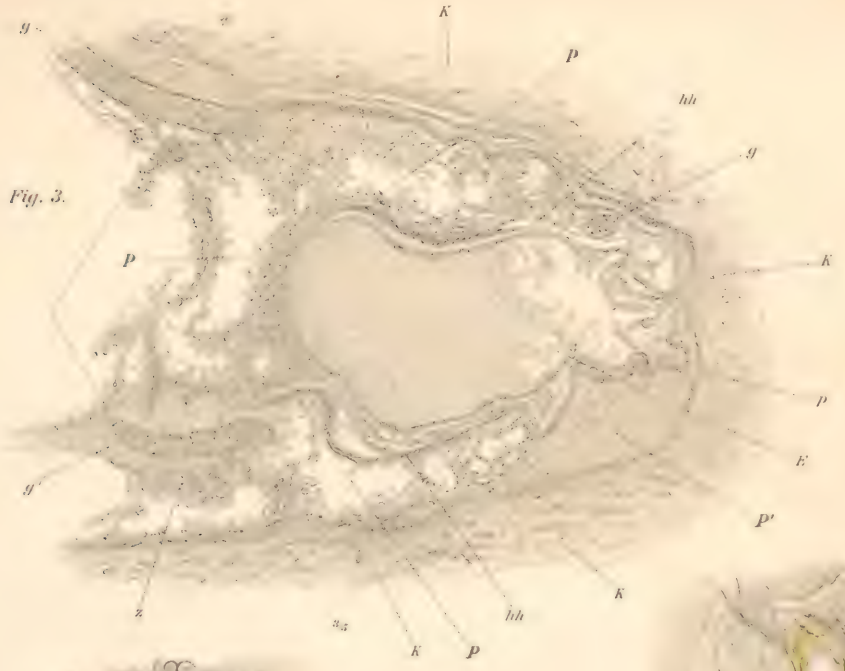


Fig. 4.

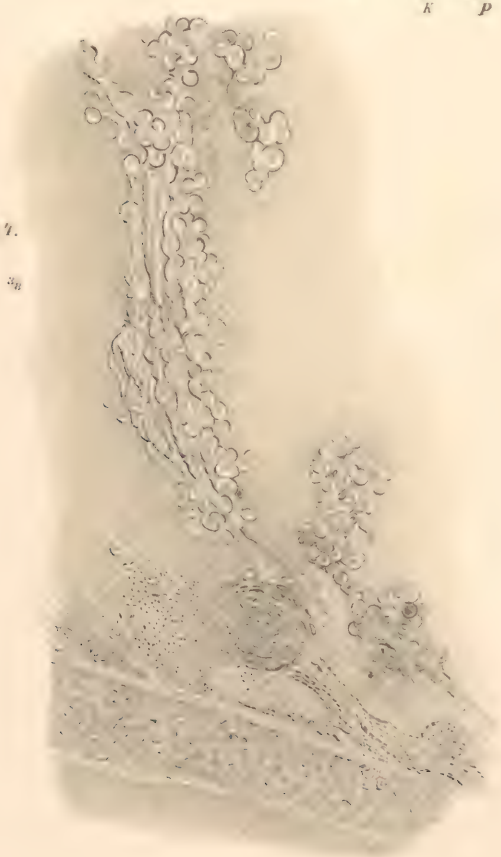


Fig. 5.

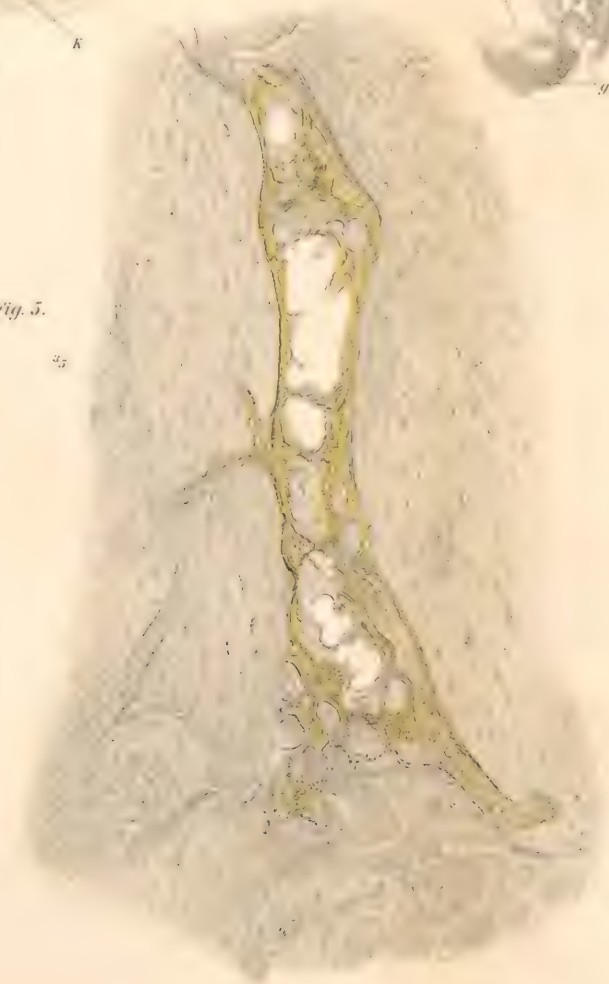


Fig. 6.

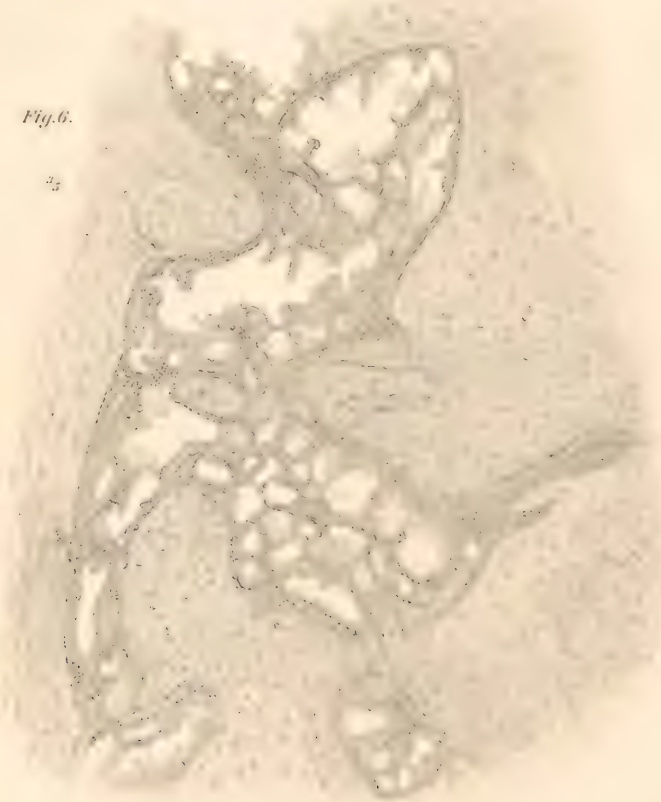


Fig. 7.

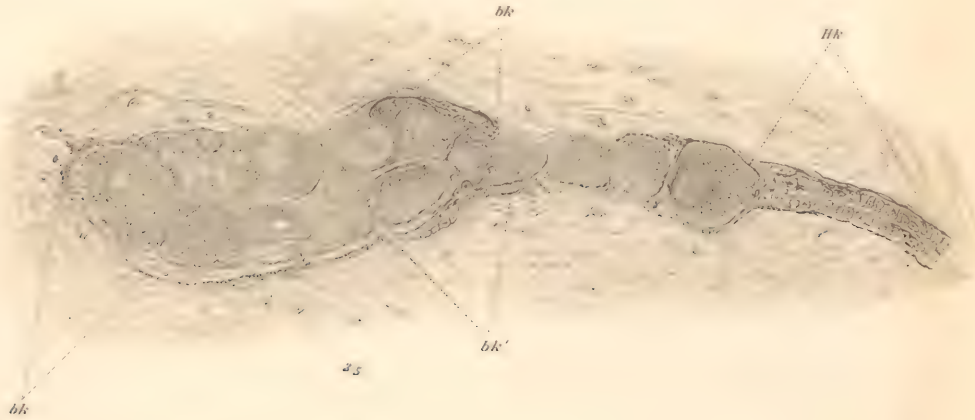

bk
Hk
bk'
bk
35

Fig. 8.

9
9'
9
9
9
8
1
9
5
3
2
19
18
17
6
9
13
14
tb
11
16
12'
tb
15
9''
7
7
Cr
12
35
9
10
10
10

Fig. 10.

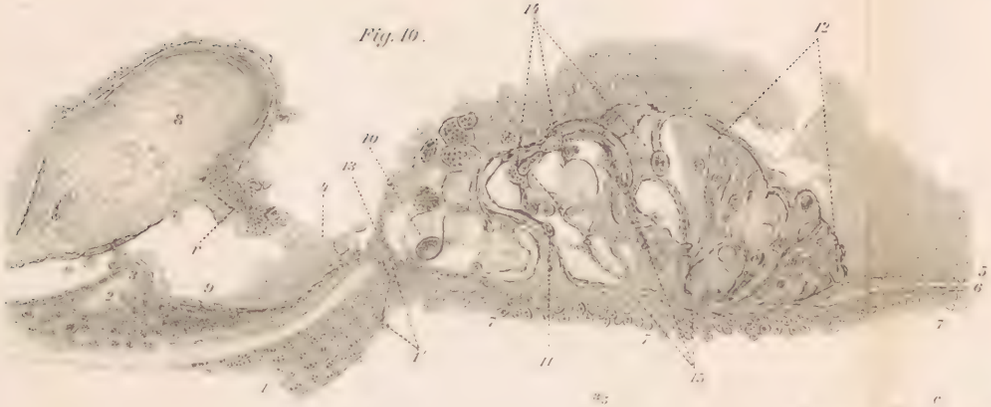
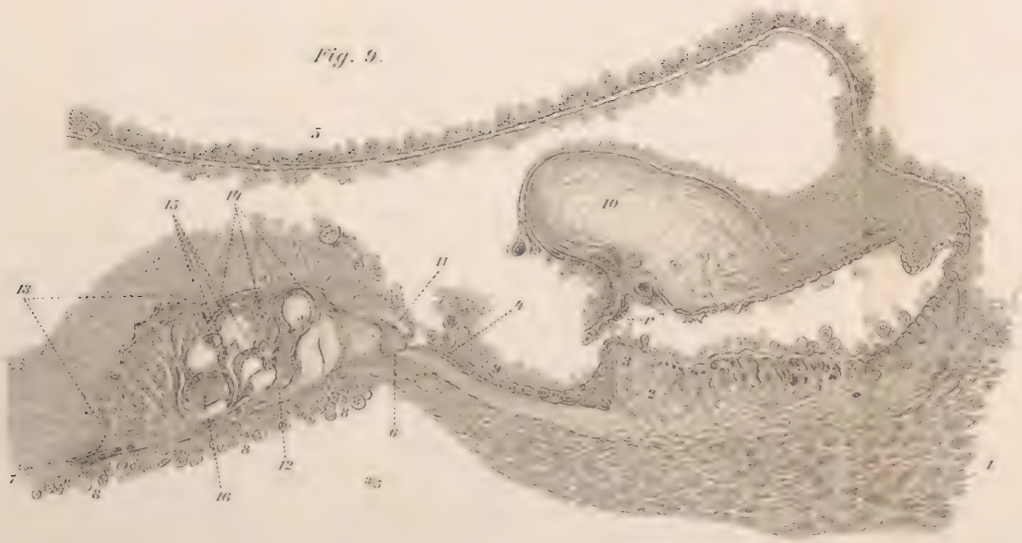


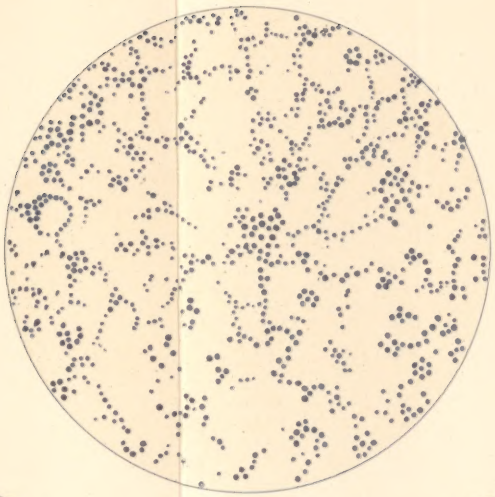
Fig. 11.

Fig. 9.



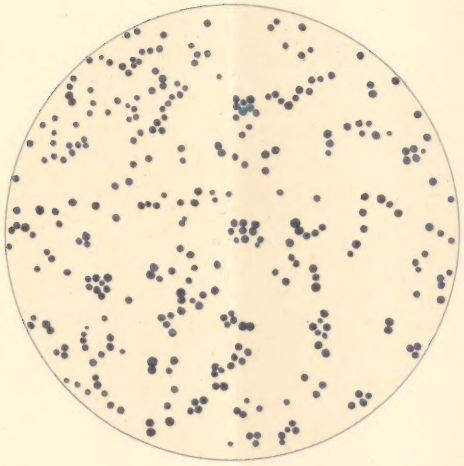
Lith. Anst. v. C. Kirst, Leipzig.

Fig. A.



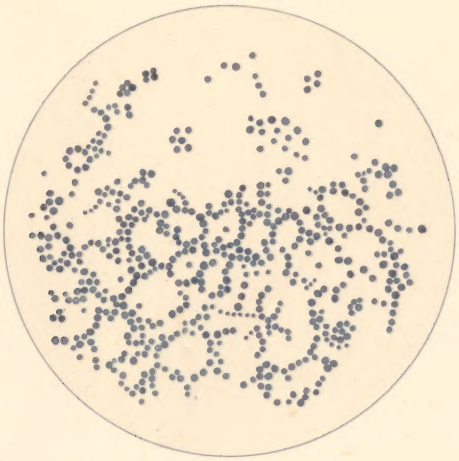
2/12

Fig. B.



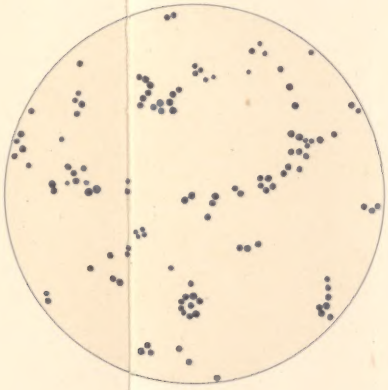
4/12

Fig. C.



4/12

Fig. D.



2/12

F. Voith, gez.

Lith. Anst. v. C. Först, Leipzig.

G. P. PUTNAM'S SONS, PRINTERS
NEW YORK